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DISTURBANCES OF THE VERTICAL MOTOR MUSCLES OF THE EYES

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I. PHYSIOLOGIC ACTION OF THE VERTICAL MOTOR MUSCLES

The disturbances occurring in the sphere of action of the vertical motor muscles of the eyes present such numerous and manifold problems to the ophthalmologist and so often give rise to unexpected and unpleasant therapeutic failures that many ophthalmologists dislike to meddle with such conditions and refer the patients to other specialists from the very beginning. There are two reasons for this. While the function of the lateral muscles of the eyes is easily comprehensible under normal and abnormal conditions and the rules for diagnosis and appropriate treatment of their disturbances may be expressed in a few sentences, the correct understanding of even the normal function of the vertical motor muscles presupposes an accurate knowledge of their anatomic and especially of their physiologic characteristics, which can be appreciated only by some one who is thoroughly familiar with the fundamental laws of the movements of the eyes. Accordingly, pathologic conditions of the vertical motor muscles are much more complicated than anomalies of the lateral muscles. This is true especially in cases in which disturbances of heterogeneous origin coexist, producing symptoms which to one without adequate clinical experience seem contradictory and present the greatest difficulties in the analysis of the disturbance and may lead to wrong diagnosis and therapeutic failures.

At the present time four separate groups of disturbances of the vertical motor muscles are known, which are entirely different with reference to their origin. Before discussing these in detail, I shall summarize briefly the most important anatomicophysiologic conditions from which the normal functions of the vertical motor muscles and their disturbances are derived.

Each eye has at its disposal two pairs of vertical motor muscles. Each pair consists of a rectus muscle and an oblique muscle, which cooperate

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both in supraversion and in infraversion. This cooperation brings about vertical movements of the eyes from the primary position without a simultaneous torsion, that is, without changing the position of the retinal meridians relative to each other. A torsional deviation takes place only in those extreme diagonal positions of the eyes which do not occur in ordinary vision because owing to compensatory movements of the head all extreme movements of the eyes are avoided. Moreover, the cooperation of a rectus muscle and an oblique elevator or depressor muscle makes it possible to maintain a certain amount of elevation and depression of the eyes in the entire field of fixation. For example, the superior rectus muscle, the muscle plane of which forms an angle of about 27 degrees with the visual line in the primary position, can effect the maximum of elevation only if the visual line is turned out by 27 degrees. If the visual line could be turned in by 63 degrees, it would be perpendicular to the muscle plane of the vertical rectus muscles and could not be elevated at all from this position, even by a maximal contraction of the superior rectus muscle. The only effect of the contraction in this case would be a torsion of the eye (inward rotation) about the visual line as axis. This holds good, analogously, for the depressing function of the inferior rectus muscle, the muscle plane of which is coincident with that of the superior rectus muscle. In other words, the vertical rectus muscles are preferably elevators and depressors of the abducted visual line, their participation in the vertical movement of the adducted eye being less the farther the visual line deviates from the muscle plane of the vertical rectus muscles. Approximately the reverse applies for the function of the oblique muscles. Since their muscle planes are vertical to the visual line when the eye is abducted by 40 degrees, the oblique muscles have absolutely no elevating or depressing effect in that position but rotate the eye solely about the visual line as axis. On the other hand, these muscles bring about purely a vertical movement when the visual line lies in their muscle plane, i. e., is adducted to the maximum (50 degrees).

Antagonistic torsional components of a rectus and of an oblique elevator (depressor) muscle compensate each other in such a way that an oblique position of the retinal meridians does not occur. Similarly, the diminution of the elevating or depressing component of one muscle during lateroversion is compensated by the accretion of the elevating or depressing function of the cooperating muscle in this very movement. This applies, mutatis mutandis, also to the increase and the decrease, respectively, of the torsional effect of those muscles in a change of the position of the eyes in the lateral direction. The action of the vertical motor muscles in the different visual directions is based on these anatomicomechanical arrangements. However, the vertical motor muscles not only are connected with each other for the purpose of executing parallel vertical movements but are also engaged, though to a considerably

lesser extent, in other combinations, in which they are governed by other centers. The elevator muscles of one eye, together with the depressor muscles of the other eye, are innervated to move the eyes in opposite vertical directions to produce positive and negative vertical divergence, just as the lateral muscles of the eyes must bring about, besides dextroversion and levoversion, the convergence and the divergence movement. In a third combination the vertical motor muscles are governed by the vestibular apparatus, and effect, when the head is tilted to one shoulder, the parallel rotary movement of the eyes toward the opposite side about the visual lines as axes. This rotary movement of the eyes requires the cooperation of both the superior muscles of one eye and both the inferior muscles of the other eye. Rotation of the eyes toward the right, which takes place when the head is tilted toward the left shoulder, is performed by the inferior rectus and inferior oblique muscles of the right eye and by the superior rectus and superior oblique muscles of the left eye, because the first pair has an outward rotating component and the latter an inward rotating component. Since the aforementioned muscles, aside from their rotating component, are antagonists, the superior rectus muscle, for example, having an elevating and adducting action and the superior oblique muscle a depressing and adducting effect, there will occur merely a torsion of the eyes about the visual lines but no change in the position of the latter, provided both muscles are innervated simultaneously and are functioning normally. The use of this parallel rotary movement for diagnostic purposes in cases of pathologic involvement of the vertical motor muscles will be discussed later. In a fourth combination the vertical motor muscles effect a rotary movement in opposite directions, namely, a conclination (intorsion) or disclination (extorsion) of the vertical meridians. The innervation required for that rotation is brought about only through corresponding fusional stimuli, for example, that presented by two identical objects which are fused in the stereoscope or the haploscope and are rotated in opposite directions. The eyes follow this displacement within certain limits, so that the images will remain on corresponding retinal areas, and binocular single vision will thus be maintained. A fifth kind of innervation of the elevator muscles must be mentioned, because it is important for diagnostic purposes. The impulse to close the lids provokes a simultaneous upward movement of the eyes (Bell's phenomenon). There are cases of oculomotor disturbances in which the eyes are not only incapable of spontaneous elevation but fail completely to react to vestibular and fusion innervations, and only the prompt execution of the elevation together with closure of the lids will show that the nuclei as well as the nerves of the elevator muscles are absolutely intact but severed from the supranuclear pathways, with the single exception of the pathway for Bell's phenomenon.

In the examination of persons with oculomotor disturbances the following fundamental laws of the movements of the eyes must always be considered:

1. All innervations which take place during the normal visual act, the spontaneous as well as the fusion innervations and those reflex-like innervations due to optic, acoustic and tactile excitations, and finally also the true reflex innervations of vestibular origin, always flow equally to those groups of muscles of both eyes which are associated in the execution of certain movements; the innervation causes at the same time the contraction of the agonists and the relaxation of the antagonists.

2. From this it follows that it is impossible to innervate one muscle or one eye alone or to send a stronger innervation to one eye than to the other. All the muscles of both eyes always participate in each movement: One half experiences an increase of tonus and the other half a decrease.

This fundamental law of ocular movements, established and formulated by E. Hering, has often been misunderstood and its strict validity doubted because equal innervation is not identical with equal movement of the eyes. If when looking at a distant object in the median plane one's attention is attracted by a near object located in the right visual line, only the left eye will make an adduction movement, and the right visual line will maintain its position unchanged. Although the movement is unilateral, the bilateral equal innervation of the eyes in such cases can be demonstrated unequivocally: Not only is there a symmetric increase of accommodation and a symmetric contraction of both pupils, but both eyes undergo the slight torsion about the visual lines as axes which is always connected with the fixation of a near object. Moreover, at the instant of the fixation of the near object it will show an apparent movement toward the right, indicating that the "double" eye has now received the impulse for dextroversion, which cannot become manifest in the right eye merely because in this eye the effect of the impulse for dextroversion is compensated by the simultaneous convergence impulse, whereas both impulses induce the left eye to move in the same direction, i. e., to the right. Against the validity of Hering's law cases have been reported in the literature in which the patients were able voluntarily to turn one eye alone either in the horizontal or in the vertical direction. I have checked up on a number of such cases and have not found any in which the unilateral movement could not be attributed to bilateral equal innervations analogous to the previously mentioned unilateral adduction movement. Voluntary unilateral divergence or vertical movements may be performed under the following conditions: There must be a horizontal or a vertical divergence, respectively, as the position of rest of the eyes, which can be kept latent by a well developed fusion mechanism. If such persons relax the

innervation correcting the anomalous position of rest, one eye will move outward and upward or downward, respectively, while the other eye remains focused on the fixation object. But if the patient will now direct his attention to the double images which have become apparent, the fusion tendency will induce the deviated eye to return to binocular fixation so that the double images will be fused. Some time ago I checked up on an especially instructive case of arbitrary unilateral vertical movements. I was able to show that the patient had an old—possibly a congenital—paresis of the trochlear nerve. He had learned to let the paralyzed eye move upward and back again to the horizontal plane, but the latter movement was accomplished only when the compulsion to fusion could become effective. If the eye in question was covered or the patient's head was tilted toward the paralyzed side, whereby a maximum deviation occurred, the squinting eye could not be brought back from its deviation. On the other hand, the patient could not turn the eye from the normal position to an upward deviation when his head was tilted toward the nonparalyzed side, thereby completely disburdening the paretic muscle. Therefore, the only voluntary factors in such cases are the disregarding as well as the cognizance of the impressions which are transmitted by one eye while the fixation with the other eye is maintained. The fusion mechanism takes care of everything else automatically.

Formerly, many a wrong diagnosis was made because in the examination of persons with disturbances of motility decisive importance was attributed to the functions of the individual muscles as they were derived from their topographicoanatomic conditions; one did not take into consideration the fact that a single muscle never executes an ocular movement separately, even if it had the power to perform the intended movement. On the basis of the fact that the isolated action of the inferior rectus muscle may bring about depression, adduction and torsion, it was assumed that there was paresis of this muscle if one eye could not move downward and inward. This assumption did not take into consideration the fact that the depressing function of the inferior rectus muscle becomes increasingly less the more the visual line is turned in. To turn the eye down and in, three muscles cooperate as agonists: the two depressor muscles and the internal rectus muscle, the principal part of the two depressor muscles being played by the superior oblique muscle, the depressing component of which grows with the increasing adduction of the visual line. A considerable restriction of the field of fixation inward and downward, therefore, points not to weakness of the inferior rectus muscle but to weakness of the superior oblique muscle. This restriction is a regular characteristic of paresis of the trochlear nerve.

II. DISTURBANCES OF THE VERTICAL MOTOR MUSCLES

The disturbances of the vertical motor muscles may be separated into the following five groups: (1) pure concomitant vertical deviations, (2) vertical deviations of paretic origin, (3) deviations that show the features of unilateral or bilateral overfunction of the inferior oblique muscle, (4) dissociated vertical deviations and (5) vertical deviations showing the combined characteristics of several of the other groups.

CONCOMITANT VERTICAL DIVERGENCE

The first group comprises the vertical deviations showing the characteristics of pure concomitant squint: Above all, the angle of squint is independent of the direction of gaze and of the transition of the fixation from one eye to the other. The diagnosis is relatively simple if one has to deal with a permanent deviation. In many cases of this type the deviation is composed of a horizontal and a vertical component; these must be examined separately in order to determine whether the vertical component is not perhaps based on an atypical secondary effect of the horizontal muscles or whether it presents the characteristics of one or more of the other forms of vertical deviation. This may be established by comparative measurement of the angle of squint in the primary position as well as in lateroversion, supraversion and infraversion and finally in diagonal positions of the eyes. This is comparatively easy if double images can be brought into the consciousness of the patient and are localized according to the normal (innate) retinal correspondence. In cases of anomalous retinal correspondence, one has to make objective measurement by means of one of the methods which are not to be discussed here. In cases in which the visual acuity of each eye is fairly equal the double images must be studied by putting the colored glass first before one eye and then before the other eye. If the results show differences in the amount and type of deviation which recur in repetitions of the test and these differences are not due to paresis, a characteristic of which is a difference between the primary and the secondary angle of squint, then these differences point to a dissociated disturbance of the vertical motor muscles. But if repeated measurements and comparison of the results show approximately the same degrees of deviation in all directions of gaze, it is fairly certain that there is a purely concomitant disturbance. In such cases the double images display no obliquity, which would point to a paretic vertical deviation and would be different in the different parts of the field of fixation. There is likewise lacking the influence of the tilting of the head to either side on the amount of the vertical divergence, which is so characteristic in many cases of vertical deviations of paretic origin.

Considerably more difficult are the examination and analysis of the latent, or only periodically manifest, vertical deviations. In such cases only a series of comparative measurements of the angle of squint will disclose with any degree of certainty the nature of the deviation. The principal difficulty is due to the fact that in such cases the innervation maintained by the fusion mechanism and compensating the vertical divergence cannot be immediately and completely relaxed when fusion is suspended. The suspension of fusion by means of the Maddox rod or a dark red glass before one eye does not suffice for the complete relaxation of the compensatory innervation which maintains an increased tonus of the elevator muscles of one eye and of the depressor muscles of the other eye any more than placing convex glasses before the eyes of a youthful subject with hyperopia induces him to give up promptly and completely the increased tonus of the ciliary muscle. Not only is relaxation caused by the breaking up of the fusion innervation incomplete, but when the patient is tested at different times it is also inconstant, so that if the test is repeated several times under the same conditions the amount of deviation will be found to be greater at one time and smaller at another. The continuous fluctuations of the compensatory innervation may occasionally simulate a paretic disturbance, since different degrees of deviation are found in the different directions of gaze. Such an error may be prevented only by repeating the measurements several times; thus an entirely dissimilar behavior of the vertical divergence may frequently be found when the subject is changing the direction of gaze. In such cases a careful determination of the vertical fusional amplitude is a valuable aid in rendering a reliable diagnosis. Take, for example, the case of a positive vertical divergence, which is usually kept latent through a negative vertical divergence innervation that causes an increased tonus of the right depressor and the left elevator muscles. If prisms of increasing strength are placed base down before the right eye, or base up before the left eye, the positive vertical divergence amplitude will be found to be considerably greater than normal, because the active positive vertical divergence innervation is required to set in only when, after the relaxation of the compensatory (negative vertical divergence) innervation, the eyes have taken their position of rest. The negative vertical divergence amplitude, on the other hand, is considerably diminished or reduced to zero, because the innervation to the negative vertical divergence must first overcome the positive vertical divergence, that is, the patient's position of rest, before it can bring the eyes into the opposite (negative) vertical divergence. With a series of tests which effect a positive vertical divergence, it is possible, as a rule, to bring about a complete, or at least a more extensive relaxation, of the compensating innervation, than by merely suspending fusion. Such examinations, of course, require time, patience and an adequate method of procedure (either a phorometer or a double

prism apparatus, a haploscope or similar apparatus). Despite every precaution and frequent repetition of the test, it is not always possible to distinguish whether one has to deal with a pure hyperphoria, i. e., an anomalous position of rest, based on anatomicomechanical factors, or whether the vertical divergence is due to abnormal innervations, which may become manifest temporarily in cases in which there is a normal position of rest as well as in those in which the anomaly of innervation is associated with a true hyperphoria. After discussion of the remaining forms of vertical deviations, I shall cite examples of such disturbances which may cause serious diagnostic and therapeutic difficulties.

PARETIC VERTICAL DIVERGENCE

Vertical deviations of paretic origin do not require detailed discussion here so far as typical cases are concerned. Whether an elevator or a depressor muscle is paralyzed is easily determined if the raising or the lowering of the eyes increases or decreases the vertical divergence. Whether a rectus or an oblique muscle is paralyzed will be decided by ascertaining whether the vertical divergence increases either in abduction or in adduction of the paretic eye and decreases in the opposite direction. Obliquity of the double images, as will be demonstrated presently, does not play a decisive role in the diagnosis. Of even less importance for the differential diagnosis is the lateral distance of the double images which may be present in addition to vertical divergence. One must not expect that in every case of paralysis of the trochlear nerve, for instance, there must be an uncrossed diplopia in addition to the vertical diplopia, owing to the loss of the abducting component of the superior oblique muscle. In 25 per cent of the cases of paralysis of the trochlear nerve either the double images show no lateral distance or they are crossed. This is due to the great frequency of exophoria as a position of rest, by which when it becomes manifest after onset of the paresis the slight convergence resulting from the absence of the abducting component of the superior oblique muscle will be compensated or overcompensated. Diagnostic difficulties may arise in a later phase of paretic vertical deviation, namely, at the time when the paretic muscle has wholly or partially regained its function, but the vertical deviation continues as a result of a secondary contracture of the antagonist of the paretic muscle. The deviation then no longer shows the characteristic signs of its paretic origin but will become more and more similar to the concomitant type. This happens rather frequently also in the atypical secondary phase of paresis of the abducens nerve, which may appear as concomitant convergent strabismus when neither a distinct motor defect nor an increase or decrease of esotropia can be ascertained in lateroversion. Paresis of the vertical motor muscles in

the atypical secondary stage shows an analogous behavior so far as the vertical divergence does not increase or decrease when the eyes are raised or lowered; thus one will not know whether an elevator muscle of one eye or a depressor muscle of the other eye had been originally paretic. But one does find regularly in such cases a considerable difference of the vertical divergence in lateroversion, according to whether the deviation is caused by a disturbance of balance between the vertical rectus muscles or between the oblique muscles. Take, for example, a case of typical paresis of the right fourth nerve in which the paresis has subsided considerably; the positive vertical divergence, however, continues, because it is maintained by the secondary contracture of the right inferior oblique muscle. It changes neither in supraversion nor in infraversion but increases considerably when the patient is looking toward the left, so that the right eye is adducted and its vertical position now depends predominantly on the oblique muscles. In dextroversion the vertical divergence recedes considerably, because the oblique muscles have no influence on the vertical position of the right eye while it is abducted. An analogous behavior of positive vertical divergence in the different directions of gaze could also be caused by paresis of the left superior rectus muscle in a later atypical stage. In such a case the vertical divergence would also attain its maximum in levoversion, during which the left eye is abducted, and its minimum in dextroversion, in which the vertical position of the left eye is influenced only in a minor degree by the vertical rectus muscles. Nor would the obliquity of the double images furnish a reliable clue as to whether the right superior oblique or the left superior rectus muscle is the cause of the vertical deviation. The obliquity merely shows whether there is disclination or conclination (extorsion or intorsion) of the vertical meridians.

Since both of the muscles under discussion are inward rotators, paresis of each of them causes a pathologic disclination. I want to emphasize the fact that it is by no means always the image seen with the paretic eye which seems oblique to the patient; quite often he will see either both images slanting or even the image of the nonparetic eye obliquely and that of the paretic eye straight; the latter results if the paretic eye happens to be the dominant eye. In such cases vertical and horizontal contours, although imaged on oblique sections of the retina (oblique meridians) of the paretic eye, will be seen vertically and horizontally; but then the corresponding images, although lying on the vertical and the horizontal meridian of the other eye, respectively, must appear obliquely, according to the angle of disparity between these meridians and the oblique meridians bearing the same images in the paretic eye. There are two possibilities for deciding which is the paretic eye in such cases. One is the head-tilting test. If the head is tilted toward the right shoulder, a vestibular innervation for a parallel rotary

movement of the eyes toward the left side will be elicited; the right eye will receive the innervation for an inward rotation and the left eye, for an outward rotation. If the balance of the right oblique muscles is disturbed, owing to paresis of the superior oblique muscle, the superior rectus muscle, when innervated together with the superior oblique muscle for an inward rotation, will produce an upward deviation of the right eye, while the left eye, in which the two lower muscles (the inferior rectus and the inferior oblique muscles) cooperate in the outward rotation, the lowering component of one muscle being compensated by the elevating component of the other, will perform merely a rotary movement about the visual line as axis. When the head is tilted toward the left, the right superior oblique muscle does not become active; the result is a decrease or complete disappearance of the vertical deviation. Since the right superior oblique muscle must participate as an inward rotator in the rotary movement to the left and the left superior rectus muscle functions as an inward rotator to the right, the results of the head-tilting test are different according to whether the vertical divergence is due to the paresis of one or the other muscle.

For an exact investigation of the influence which the position of the head exerts in certain cases of vertical deviation, one may use a simple apparatus constructed on the principles of the Helmholtz *Visierzeichen* (fig. 1). While the patient's head is fixed by taking the little plate at one end of the rod between his teeth, he looks at a horizontal black strip on a piece of white cardboard fixed to the other end of the rod 30 inches (75 cm.) away. The rod is put through a short tube, so that when the patient tilts his head it rotates around the same axis and through the same angle as the head. This insures the maintenance of the direction of gaze during the tilting of the head, since the cardboard with the fixed strip keeps pace with the movement of the head in respect to both the amount and the direction.

Another method which may be used to determine whether the vertical deviation is due to paresis of the oblique depressor muscle of one eye or to the elevator rectus muscle of the other eye is as follows: When one looks in a dark room at a faintly glowing filament, viewed monocularly through a tube, it will as a rule appear vertical and horizontal, respectively, to the observer only when its image lies on the vertical and the horizontal meridian, respectively, of the retina concerned. In paresis of a muscle with a rotating component, these meridians are positioned obliquely; consequently the glowing filament must be given a correspondingly oblique position in order to appear vertical or horizontal to the paretic eye of the patient. If the two eyes, which have been tested consecutively, show essential differences in the localization of the vertical and the horizontal meridian, one must derive therefrom to which eye the paretic elevator or depressor muscle belongs.

OVERFUNCTION OF THE INFERIOR OBLIQUE MUSCLES

The third group of disturbances of the vertical motor muscles is closely related to the atypical vertical deviations of paretic origin just discussed but is nevertheless distinguished from the latter by certain peculiarities. In its purest and simplest form, this type of disturbance presents the following characteristics. When the patient is looking straight ahead the visual lines are parallel, and even when the compul-

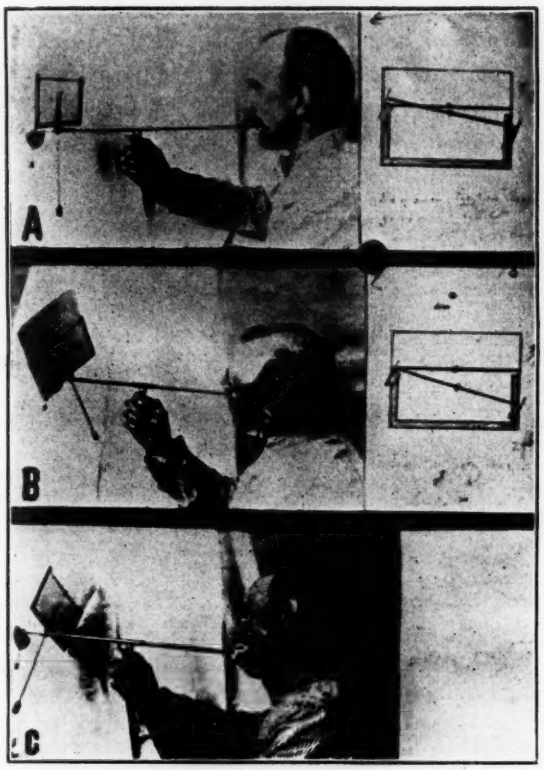

Fig. 1.—The head-tilting test as demonstrated by patients with palsy of the left trochlear nerve. *A*, two images of the fixed black strip are seen (reproduced on the right side of the photograph) while the patient's head is erect. In *B*, the vertical distance between the two images is increased by tilting the head toward the left shoulder. In *C*, the tilting of the head toward the right shoulder helps to secure binocular single vision.

sion to fusion is eliminated they show no, or only unimportant, latent disturbances of balance. But if the eyes follow an object moving laterally in the horizontal plane, the adverting visual line will deviate upward, occasionally by a considerable amount (fig. 2). If the disturbance affects both eyes, the left eye will deviate upward in dextroversion, and the right eye will deviate upward in levoversion. When the fields of

fixation of either eye are tested, it will be seen that each field is considerably enlarged in the upper nasal quadrant without being restricted in the upper temporal quadrant. The limits of the lower halves of the field of fixation are either normal or slightly extended in the lower temporal quadrant. The behavior of the double images corresponds to the objective findings. Their obliquity points to a disclination of the vertical meridians; it is absent in some cases of congenital origin. The head-tilting test sometimes gives negative results; occasionally, however, in cases in which the deviation is manifest only when the patient is looking toward one side the results are positive, since the test shows a disturbance analogous to the atypical secondary stage of paresis of the trochlear nerve discussed previously. Frequently such an upward squint of the adducted eye during lateroversion will be found combined

Fig. 2.—*A*, patient with binocular fixation in the primary position of the eyes. In levoversion the right eye deviates upward, as in *B*. In dextroversion the left eye deviates upward, as in *C*.

with either convergent or divergent strabismus; in the primary position of the eyes no vertical deviation, or only a minor one, is to be found in addition to the horizontal deviation (figs. 3 and 4).

The anomaly of the vertical motor muscles just discussed is either unilateral or bilateral. It shows the features of an overfunction of the inferior oblique muscles, because of its far reaching conformity to the atypical secondary phase of paresis of the trochlear nerve, in which only the overfunction of the inferior oblique muscle, owing to a secondary contracture, may cause the deviation. But a primary overfunction of the inferior oblique muscle certainly does occur. I have seen it arise after radical operations on the frontal sinus, although paresis of the superior oblique muscle is found much more frequently in these cases. If in such operations the trochlea is not brought back

to its right place through an exact suture of the periosteum but remains dislocated, the function of the oblique muscles must be disturbed, at least temporarily. Usually there is a weakening of the function of the superior oblique muscle, but occasionally the function of the inferior oblique muscle is increased even without the weakening of its antagonist. This is explained by the fact that the action of the inferior oblique muscle is checked by a fascial ligament which is inserted in the trochlea. Its influence would be either lost or reduced if the trochlea were dislocated or the ligament itself were injured. C. H. Sattler also reported, after me, on the primary overfunction of the inferior oblique muscle after operations on the frontal sinus. In such cases the genesis and nature of the anomaly are vouched for. While the analogy of the

Fig. 3.—*A*, a patient with convergent strabismus of the right eye. The left eye is looking straight ahead. In dextroversion the left eye goes up while the right visual line remains in the horizontal plane, as in *B*. In levoversion the right eye goes up while the left eye is turned out, as in *C*.

symptoms in the much more numerous cases in which the anomaly is function of the inferior oblique muscle, there are no reliable data concerning the genesis, and especially concerning the fact whether it is a primary or a secondary disturbance, i. e., whether or not it developed in the course of a paresis of the trochlear nerve which existed originally but was cured. It might also be conceivable that the upward movement occurring simultaneously with the adduction of the right eye in a lateroversion impulse may be due to an asymmetric (oblique) insertion of the internal rectus muscle, as was mentioned by Cords on the strength of several cases in which he operated. In the numerous cases of apparently congenital overfunction of the inferior oblique muscle in which

the anomaly of the muscle was accompanied by convergent strabismus, I have found only occasionally a slight asymmetry of the insertion of the internal rectus muscle, so that it could not be held responsible for the extreme upward movement of the eye during the inward movement.

Fig. 4.—*A*, patient with divergent strabismus of the left eye. The right eye is looking straight ahead. In dextroversion the left eye goes up while the right visual line remains in the horizontal plane, as in *B*. In levoversion the right eye goes up while the left visual line remains in the horizontal plane, as in *C*.

In some cases the absence of certain characteristics which are never lacking in a true overfunction of the inferior oblique muscle argues in favor of the possibility that the type of overfunction of the inferior oblique muscle noted may be caused by other factors. That is to say, while there is always a meridional disclination in cases in which the

balance of the oblique muscles is disturbed by overfunction of one muscle or a weakness of the other, the disclination is surely absent in some cases of apparently primary overfunction of the inferior oblique muscle and there is likewise lacking the influence of the head tilting on the amount of the vertical divergence. Moreover, the position of the eyes when the subject is looking straight ahead is often perfectly normal in cases of primary overfunction of the inferior oblique muscle; this is never found in cases of secondary contracture of the inferior oblique muscle in which the contracture was preceded by paralysis of the antagonist. Aside from the few instances in which an oblique insertion of the internal rectus muscle may lack the aforementioned characteristics of a secondary anomaly, it is not yet known what is at the bottom of the primary overfunction of the inferior oblique muscle. On the other hand, there are many cases of apparently primary overfunction which bear all the essential characteristics of the aforementioned overfunction. This applies particularly to the congenital disturbances of this type, i. e., those already observed in earliest childhood and often brought to the attention of the physician because of the habitual torticollis existing since the second or the third year of the child's life. Of course in such cases the secondary, i. e., the postparetic, nature of the muscular disturbance cannot be excluded.

For therapy it is fairly unimportant whether overfunction is primary or secondary. What is important is merely the proof that one has to deal with such an overfunction, for example, of the right inferior oblique muscle, and not with a paretic weakness of the left superior rectus muscle. The latter is often assumed erroneously in cases of this kind, because when one looks up and to the left the left eye lags behind the right. If, as is often the case, the right eye is the dominant eye, its motility is decisive for the innervation of the two eyes. And when the function of its inferior oblique muscle is increased abnormally, the right eye will require a much smaller innervation to reach the limits of the upper nasal quadrant of the field of fixation than is required by the left eye with the normal superior rectus muscle in order to reach the normal limit of the upper temporal quadrant with a parallel movement. But if the fields of fixation of the individual eyes are examined separately, one eye will show no constriction of the field outward and upward, but there will be considerable extension of the field of the other eye when one looks up and inward. Moreover, in the majority of cases under discussion the increase of the vertical divergence when the head is tilted toward one side and the decrease of the vertical divergence when it is tilted toward the other side are decisive for the diagnosis of a disturbance of balance among the oblique muscles. The decision is important for the therapeutic procedure. The weakening of the excessively strong inferior oblique muscle through myectomy is considerably simpler and

more amenable to treatment than the strengthening of the function of the superior rectus muscle of the other eye by advancement or resection, which, in addition, has the disadvantage that as a rule it leaves the patient with a cosmetically disfiguring enophthalmos and slight ptosis. I have obtained most gratifying results in many cases with myectomy of the inferior oblique muscle. After binocular vision had been made possible with the head in the normal position, the habitual torticollis eventually disappeared, as did also the disfiguring upward squint when the eyes looked sideways. When the vertical divergence was associated with a considerable divergent or convergent strabismus, I found it expedient to remove first the lateral deviating component, after which, occasionally, a further operation became unnecessary. If the vertical divergence is also present in the lower half of the field of fixation, myectomy of the inferior oblique muscle may occasionally not remove it in this field, although it will remove the vertical divergence in the horizontal and the upper field. In such cases the muscular balance of the eyes can be restored by careful retroplacement of the inferior rectus muscle of the other eye, on the strength of the same deliberations which are decisive for using this operation in cases of inveterate palsy of the trochlear nerve.

DISSOCIATED VERTICAL DIVERGENCE

The most interesting disturbances of the vertical motor muscles, because their genesis and symptomatology are so fundamentally different from those of the disturbances so far discussed, are the so-called dissociated vertical deviations. Their peculiarities are most marked in cases of alternating or double hyperphoria, as is shown by the patient in figure 5. Ordinarily he had binocular fixation. Only occasionally, when he was tired or when he gazed abstractedly ahead, one or the other eye went up. With the diplopia test the image of the right eye at one time and that of the left eye at another time would be seen below the other one, according to whether the red glass was held before the right eye or the left. The characteristic difference between such a vertical deviation and those discussed previously is obvious. These deviations are either concomitant or of paretic origin, or they are due to an overfunction of one of the vertical motor muscles. The joint feature of all these deviations is that when the eye which had been deviated upward was directed to the object which was first fixated by the other eye, the latter deviated downward. This alternation between upward and downward squint, which is caused by the transition of fixation from one eye to the other, is based on the law of the equal innervation of both eyes. To look at an object lying in the horizontal plane with the upward squinting eye, an infraversion impulse is called

for, which makes the eye which had first been in a horizontal position deviate downward. In contrast to this behavior, in the cases now under discussion, if the eye which is deviated upward is induced, by covering the other eye, to focus on the fixation object, the covered eye will first make a slight downward movement accompanying the parallel movement of the other eye, but immediately after the latter has become fixed an isolated upward movement of the covered eye will take place. Such anomalies were previously attributed to a mechanical superiority of the elevator muscles over the depressor muscles. This conception is erroneous. If the assumed superiority of the elevator muscles were the same in both eyes, as for example in cases of associated paresis of the depressor muscles, the eyes would be brought to a horizontal position

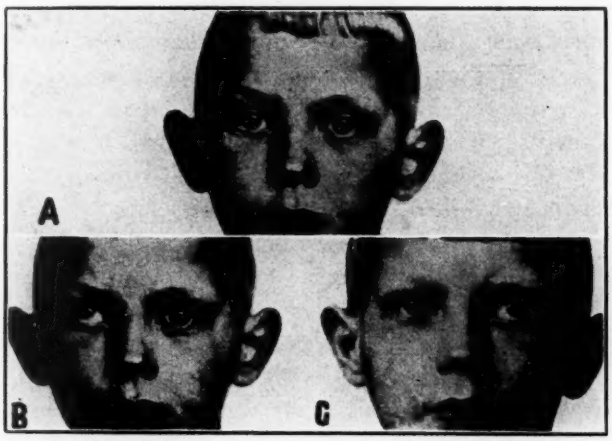

Fig. 5.—Patient with alternating hyperphoria. The visual lines either are parallel (*A*) or are deviated upward alternately (*B* and *C*).

by an increased infraversion impulse. An alternating upward deviation would not take place. And if the superiority of the elevator muscles were different on the two sides, for example, if the right depressor muscles were weaker than the left ones, the right eye with the weaker depressor muscles would be deviated upward, and if it were brought to the horizontal position by a depression impulse, the latter would bring the other eye into a downward deviation. It is easy to prove that the hypothesis of a superiority of the elevator muscle is untenable. If in a case of alternating hyperphoria, while the right eye is fixing a light located in the horizontal plane and the left eye being deviated upward, a darkening glass is held before the fixing eye, an isolated downward movement of the upward squinting eye will take place which will bring it down to, or even below, the horizontal position, while the fixing eye maintains its position unaltered. This one-sided vertical movement of

the deviated eye can be observed frequently, even without the darkening of the fixating eye. While this eye maintains constant fixation, the other eye, which is deviated upward behind a screen, will make at irregular intervals vertical movements of different extent, so that at one time a greater degree of upward deviation may be observed and at another time there may be a lesser degree of upward deviation or at times even a slight downward deviation. Such behavior, of course, cannot be reconciled with the assumption of an elastic superiority of the elevator muscles or of paretic disturbances among the elevator and depressor muscles. The alternating hyperphoria and the aforementioned one-sided vertical movements must be attributed to intermittent and alternating innervations for positive and negative vertical divergence, which are independent of the will and fluctuate in intensity. The ascertainment that in cases of alternating hyperphoria positive and negative vertical divergence movements may be elicited to more than an average extent by adequate fusion stimuli argues in favor of this assumption. It is known that normally not more than 3 or 4 prism diopters may be overcome by a vertical divergence innervation; furthermore, it is known that in concomitant hyperphoria the amplitude of either positive or negative vertical divergence is enlarged, whereas the opposite movement is either restricted or zero, according to whether it is a right or a left hyperphoria. In alternating hyperphoria, however, vertical prisms of 10 prism diopters and more in either direction may be overcome by corresponding vertical divergence innervations. This behavior refutes most impressively the hypothesis of a bilateral superiority of the elevator muscles over the depressor muscles as the basis of alternating hyperphoria. The increased amplitude of both positive and negative vertical divergence which manifests itself in the capacity to overcome unusually strong prisms in front of either eye, base down as well as base up, can be due only to the fact that with this test the one-sided vertical movements are brought about by positive and negative vertical divergence innervations. The movement is one-sided, because, as was discussed previously, an innervation to a parallel vertical movement caused by the fixation effort is combined with the fusion innervation which drives the two eyes in opposite directions, the latter compensating the effect of the former innervation in one eye while increasing it in the other eye.

Where these abnormal excitations originate is still entirely obscure. Only the following fact is known. The fusion mechanism, so long as its action is normal, keeps the tendency to alternating hyperphoria latent, just as it does in the cases of ordinary heterophoria. If, however, the fusion mechanism is weakened, for instance when fatigue sets in, or is lost entirely owing to blindness or amblyopia of one eye, the abnormal vertical divergence innervation becomes manifest and produces either alternating hyperphoria or in cases of unilateral amblyopia one-sided,

irregular upward and downward movements of the amblyopic eye. In cases in which there are no complications the direction of gaze has no influence on the anomaly under discussion.

This anomaly is often combined with concomitant squint (fig. 6). The squinting eye then shows in addition to the lateral deviation component an inconstant vertical deviation component, the extent and direction of which may be influenced, to a certain degree, by the occlusion of the squinting eye as well as by darkening the fixing eye by a colored glass. Such cases have been misinterpreted in various respects. It was believed that the isolated upward deviation of the squinting eye when it is covered and its isolated downward movement when it is uncovered had to be considered as a fusion movement, although the isolated movement of the squinting eye did not do away with the horizontal deviation. It was assumed that a binocular visual act existed on the basis of an

Fig. 6.—*A*, patient with convergent strabismus of the right eye. This is the same patient shown in figure 3. In *B*, the right eye is turned up and in, the left eye remaining in its primary position.

anomalous retinal correspondence adapted to the horizontal deviation of the squinting eye and that, owing to a subsequent change of the anatomic position of rest, a vertical deviation had arisen in addition to the horizontal deviation, but had been kept latent during vision with both eyes. More thorough investigations refuted this assumption. It could be shown that the downward movement of the squinting eye took place also when the fixing eye was darkened by a colored glass, although the squinting eye remained covered. This, of course, precluded the influence of the binocular effect of a fusion innervation. One was forced to the same conclusion by the observation that even in cases of extreme amblyopia of the squinting eye which precluded the possibility of a fusion innervation, covering the squinting eye brought about or expedited its upward movement; uncovering the squinting eye or darkening the fixing eye caused the isolated downward movement of the former. Some authors interpreted the isolated downward movement of the squinting eye when the fixing eye was darkened

as a movement connected with the reaction of the pupils to light, due to an abnormal extension of the nervous excitation in the nuclear region. If that were actually the case, then the pupillary dilatation, occurring not only during the darkening of the fixing as well as the squinting eye but being greatest when both eyes are darkened, would have to bring about the one-sided downward movement. That never happens. The darkening of the amblyopic eye has the opposite effect of darkening the fixing eye: The former effects or expedites the isolated upward movement of the squinting eye and the latter effects or expedites the isolated downward movement, while a uniform darkening of both eyes, which causes a still greater dilatation of the pupils, is followed by no change whatever in the deviation of the squinting eye or only by those slight vertical movements which may also be observed when both eyes are uncovered. Moreover, I was able to prove that the one-sided vertical movements also take place when a restriction of the pupils, resulting from a stronger illumination, is brought about instead of a dilatation. In place of the covering screen I brought a mirror into the median plane of the head, rotating it about its vertical axis until it barred the fixated object from the amblyopic eye and at the same time exposing it to a stronger light by reflecting into the eye the light from a window. In spite of the bilateral pupillary constriction which this produced, the same upward movement of the squinting eye took place as when the squinting eye was covered by a screen, which brought about a pupillary dilatation. This, and other tests, made it likely that an abnormal excitability of the centers controlling the antagonistic vertical movements remains latent so long as both retinas receive homogeneous excitations, while the one-sided weakening or exclusion of the sensorial impressions makes the excitation of that vertical divergence center manifest, which causes the upward deviation of the eye concerned or the downward deviation of the other eye, according as the fixation impulse occurs in the one or the other. That the one-sided movements of amblyopic eyes are based on the same innervational anomaly as alternating hyperphoria is evident from the fact that when the amblyopic squinting eye is covered it goes upward; when it is uncovered, or the fixing eye is darkened, it goes downward—the same behavior as is observed in cases of alternating hyperphoria when one eye is covered or the other is darkened. The upward squint of the better eye in cases of unilateral squint cannot, of course, be demonstrated if the squinting eye has no central fixation.

If attention is paid to possible dissociated disturbances of the vertical motor muscles during examination of persons with heterophoria and strabismus, one will be surprised how frequently they occur combined with concomitant deviations. I have been able to ascertain such a combination in more than 41 per cent of all cases of congenital or acquired amblyopia of one eye and am convinced that the tendency to dissociated

vertical movements exists even in persons with binocular vision much more frequently than one would suppose; only, it is a great deal more difficult to demonstrate the disturbance so long as it is kept latent by the compulsion to fusion.

The practical and clinical importance of the dissociated vertical deviations is, first of all, that they do not lend themselves to any optical or operative therapy such as would be considered in cases of concomitant or paretic deviations. I have seen cases of dissociated vertical deviations in which, owing to misinterpretation of their peculiarity, tenotomy of both superior rectus muscles had been performed. Thus the parallelism of the vertical meridians of the retinas, which in all probability existed previously, was transformed into a disclination, which expressed itself in that contours were seen in double images crossing each other at acute angles. The alternating hyperphoria which manifested itself temporarily and alternately continued and differed from the primary condition only so far as the vertical movements issued from a lower position of rest, and, although they did not extend so far upward, the downward extension was correspondingly greater. The only possible way of reducing or checking these deviations is through strengthening or relieving the fusion innervation. The stronger the fusion mechanism, the longer and the more easily it will keep the tendency to dissociated vertical movements latent. I have reported on the operative results in cases of divergent squint in which the squint was combined with vertical deviations of the dissociated type. After binocular single vision had been restored by correcting the lateral deviation, the formerly marked dissociated vertical movements disappeared completely and could no longer be elicited, even by a darkening of one eye. It goes without saying that in cases of unilateral amblyopia in which the amblyopia cannot be corrected, the dissociated anomaly continues even after the correction of the coexisting lateral deviation.

Alternating hyperphoria is much rarer than the combination of the dissociated anomaly with the other disturbances of muscular balance of paretic and nonparetic origin. Figure 7 shows a patient with paralytic divergence combined with dissociated vertical squint. The right eye is amblyopic and demonstrates alternately pure divergence, divergence and upward deviation and divergence and downward deviation. The vertical upward and downward deviations of the right eye are added at irregular intervals to the stationary divergence, but they may also be elicited by covering the right eye or darkening the left eye.

The complication of all kinds of squint with dissociated anomalies may, occasionally, render the analysis of the cases difficult. This applies especially to cases in which the dissociated disturbance is combined with a deviation of the concomitant or paretic type. Wrong diagnoses may be avoided by making systematic tests of the kind and amount of

the vertical deviation during fixation with the right and the left eye in all parts of the field of fixation. It may happen, for example, that in a case of paralysis of the right trochlear nerve it will be found that the right eye is considerably higher when the red glass is placed before the right eye. If it is placed before the left eye, there will be found either no vertical deviation or even a slight negative vertical divergence. I have seen such cases in which the diagnosis of paralysis of the

Fig. 7.—*A*, patient with divergent paralytic strabismus of the right eye. In *B* the right eye is deviated up and out. In *C*, the right eye is deviated down and out.

trochlear nerve was established unequivocally on the strength of other signs and in which the peculiar behavior of the double images when tested with the dark red glass became intelligible only after I had recognized the complication of the paralysis with a dissociated disturbance of the vertical muscles. In the case of paresis of the right trochlear nerve, if the red glass is placed before the right eye the positive vertical divergence decreases because the dissociated component is added to the paretic component. But if the red glass is placed before the left eye, the paretic positive vertical divergence is

either partially or completely compensated or even overcompensated by the dissociated upward movement of the left eye, owing to the darkening of that eye.

What I have been able to say here concerning the peculiar dissociated disturbances of the vertical motor muscles is, of course, merely an outline of their most important and, in the majority of typical cases, regularly recurring symptoms. For many details which in some cases render the picture even more diversified, I must refer to my article on the dissociated vertical movements (1931).[1]

III. DIFFICULTIES IN DIFFERENTIAL DIAGNOSIS

In conclusion, I should like to make just a few remarks concerning the difficulties often encountered in separating the two groups of disturbances of the vertical motor muscles according to their different origin. One group comprises the concomitant deviations and those of paretic origin, including the cases in which there is an overfunction of the inferior oblique muscles. The second group comprises the deviations based on dissociated vertical divergence innervations. Their principal characteristic is the great instability of the deviation in contrast to the relatively great stability of the vertical divergence of the first group. But there are many exceptions even to the principal characteristic. In the discussion of concomitant hyperphoria I have already pointed out that the compensatory innervation maintained by the compulsion to fusion does not relax completely when the latter is suspended, but only partially, and in the individual investigations the relaxation is greater at one time and less at another. The concomitant nature of the deviation may be recognized and its spastic origin probably excluded by a careful measurement of the fusional amplitude, through which an extensive relaxation of the compensating innervation may be achieved. But the more thoroughly one examines the individual patients with motor disturbances and the more experience one gains in this field, the more one is convinced that frequently anomalies of the position of rest or paretic disturbances are complicated by abnormal excitations of the centers controlling the nonparallel movements. It is a well known fact that convergent strabismus represents only exceptionally a pure anomaly of the position of rest but is caused to a greater or lesser extent by an increased tonus of the convergence innervation. It is known how difficult it is to separate these two etiologic components and thus to create the reliable foundation for a rational therapy. Far less familiar is the combination of spasms of vertical divergence with deviations apparently based on an anomalous position of rest. The nonrecognition of the

1. Bielschowsky, A.: Die einseitigen und gegensinnigen ("dissozierten") Vertkalbewegungen der Augen, Arch. f. Ophth. **125**:493, 1931.

nervous (spastic) component may cause entirely unexpected operative results if not complete failure.

I should like to illustrate this with the following examples:

CASE 1.—A patient suffered for many years from severe headaches and, as he called it, "unsteady vision," which apparently was due to diplopia. I found a negative vertical divergence of 9 degrees and made a careful recession of the left superior rectus muscle, with a safe-guarding suture. Four days later there remained only a minimal overeffect in the form of a positive vertical divergence of 1 degree. In the course of the next day this increased to 9 degrees. I felt obliged to advance the retroplaced left superior rectus muscle. For eight days the result was excellent: Not a trace of the vertical divergence was demonstrable. Three days later the patient returned and complained of occasional diplopia, which he could overcome, but not without getting severe headaches. Examination showed a negative vertical divergence of almost the same amount as had been present before the beginning of the treatment. I sent the patient, who had a well developed neuropathic constitution, to the mountains for four weeks. When he came home he had no further complaints and no vertical divergence; nor did the vertical divergence return in later years. I shall postpone the epicrisis of this case until the other cases have been discussed.

CASE 2.—A 27 year old engineer, intelligent, but with a severe neuropathic constitution complained of headaches and a most annoying feeling of sluggishness (dulness), which paralyzed his capacity to work. I found a greatly fluctuating positive vertical divergence. After relaxation of the compensating innervation, an apparently pure concomitant positive vertical divergence of 15 (arc) degrees became manifest. The recession of the right superior rectus muscle reduced the vertical divergence to 1 degree, but in the course of the next two weeks it increased again to approximately its former level. After a prolonged period of observation, during which there was no important change, I decided, on the strength of deliberations into which I need not enter at this time, to perform a recession of the left inferior rectus muscle. The immediate result was a slight overeffect (negative vertical divergence), which increased in about a week to 12 degrees. Since I had been anxious to graduate the effect of the recession carefully, I could not convince myself that the negative vertical divergence was the result of an excessive weakening of the left inferior rectus muscle. My doubts proved to be justified when I had both eyes of the patient bandaged for several days and then examined him again. I found an almost complete vertical balance, the position of the eyes fluctuating between 1 degree of positive vertical divergence and 1 degree of negative vertical divergence. After the patient had his eyes uncovered during the day, he again showed 12 degrees of negative vertical divergence in the evening. Nevertheless, he was able to bring about at the double prism apparatus 7 degrees of positive vertical divergence without the least difficulty, which seems inconsistent with the assumption that the 12 degrees of negative vertical divergence which he had demonstrated formerly with fusion suspended had to be attributed to the weakening of the left inferior rectus muscle. I discharged him after giving him a weak prismatic spectacle and directions for fusion exercises. He felt well for several months, but finally returned, because his condition had become considerably worse. The negative vertical divergence amounted to 14 degrees and appeared to be of a purely concomitant nature. The advancement of the left inferior rectus muscle resulted in a positive vertical divergence of 10 degrees, which still continued ten days later—an unusually powerful effect of a simple advancement. Two weeks later a negative vertical divergence of 6 degrees was

again found, which, however, already changed during the examination and the constant exclusion of the fusion tendency to a positive vertical divergence of 2 degrees. In the further course the eyes always oscillated by several degrees about the normal position in one direction or another; finally, the diplopia disappeared, and the patient was free from complaints and has remained so.

An analogous behavior could be observed in a third case.

CASE 3.—A 45 year old man complained of diplopia, which presented no paretic characteristics but an extensive instability. The maximum positive vertical divergence which could finally be determined amounted to 18 degrees. After recession of the right superior rectus muscle there was absolute balance of the vertical motor muscles for five days. On the sixth day there was a negative vertical divergence of 10 degrees, which did not increase during elevation of the eyes, as would be expected in a case of weakness of the right superior rectus muscle, and even decreased temporarily to zero when fusion was excluded permanently. I had the patient under observation for seven months and found, with the same method of examination, 25 degrees of negative vertical divergence at one time and none whatever at another time. However, the patient's discomfort finally induced me to make a careful advancement of the right superior rectus muscle. Within the first week after the operation the slight overeffect, which is customary in an advancement, was reduced to 2 degrees of positive vertical divergence. In the second week it rose to 18 degrees. On no other occasion have I ever found that the mechanical effect of a simple advancement accomplished a change of position of approximately 43 (arc) degrees, from 25 degrees of negative vertical divergence to 18 degrees of positive vertical divergence. The fact that this enormous effect developed only during the second week after the operation is just as extraordinary, because the effect of an advancement is always greatest directly after the operation and then decreases. Evidence that the excessive effect of the advancement was not due to mechanical causes, i. e., to the change in the mechanical conditions of the position of the eyes, could be adduced from the fact that during the examination of the vertical fusional amplitude an absolutely identical and abnormally high positive and negative fusional amplitude was found. The patient was able to overcome 16 prism diopters base up or base down before one eye as well as before the other. It may be noted that in a few months all his troubles disappeared without recourse to any further measures, and after five years he showed only a slight fraction of his original vertical divergence.

I believe that the behavior in these cases can be interpreted only in one manner; that is, that the vertical divergence was due, at least to a great extent, to spasms first of one and then of the other vertical divergence innervation, which I had not been able to ascertain before the treatment or which possibly developed only in the course of the therapeutic procedure. All 3 patients had the following signs in common: The high amount of postoperative deviation existed only when both eyes were used and disappeared when one or both eyes were bandaged, which, of course, excluded all innervations produced by the fusion mechanism. Moreover, contrary to heterophorias of concomitant or paretic origin, both antagonistic innervations were increased beyond the norm and, what is more, the increase was approximately identical. The marked neuropathic constitution of the patients further supports

the assumption of the spastic nature of the temporary increase of the vertical divergence, which cannot be explained by mechanical factors.

I must confine myself here to this outline without entering into the details of the analysis. The foregoing observations warrant merely the issuance of the warning that in cases of vertical deviations one should always bear in mind the possible complications with spastic vertical divergence innervations and try to follow their signs in order to prevent unexpected and undesired operative results.

PERIMETRIC STUDIES IN SYPHILITIC OPTIC NEUROPATHIES

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A study of syphilitic optic neuropathies was undertaken in 1931 jointly by the division of syphilology of the medical clinic and the department of ophthalmology of the Johns Hopkins Hospital and University. This investigation included repeated special clinical and ophthalmologic studies on a large group of syphilitic patients over a period of years, repeated perimetric studies and determinations of light sense, and a comparison of the effects of various types of treatment on primary syphilitic atrophy of the optic nerve. There are available for correlation, therefore, accurate clinical records and data on the visual fields of a large number of syphilitic patients with disturbances of the optic neuro-pathways who were followed for years during the progression or regression of the disease. This paper is a report of the changes in the visual fields occurring in association with syphilitic optic neuropathies.

HISTORICAL SURVEY

PERIMETRIC FINDINGS IN CASES OF PRIMARY ATROPHY OF THE OPTIC NERVE

In 1903 Uhthoff [1] summarized the results of his own and other investigators' perimetric studies in cases of tabetic atrophy of the optic nerve. He concluded that, while there was no defect in the field absolutely typical of syphilitic atrophy of the optic nerve, two general types could be recognized. The first type was characterized by a more or less equal reduction of form and color sense and of peripheral and central acuity. The capacity to perceive red and green was lost early; the perception of blue and yellow was retained somewhat longer. This

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1. Uhthoff, W.: Die Augenveränderungen bei den Erkrankungen des Nervensystems, in Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1909, vol. 9, pt. 2, chap. 22, p. 192.

type of defect was considered by Uhthoff to be due to an involvement of the entire cross section of the nerve. In the second type the defective regions of the field were sharply delimited from normally functioning regions. The changes consisted in sector-shaped defects or marked concentric contraction with good visual acuity and color sense, indicating a complete sparing of the nerve fibers supplying the central portion of the retina. The sector-shaped defects might or might not be connected with the blindspot. Defects starting as central scotomas were included in this group. Central scotoma occurred in only 2 per cent of cases in Uhthoff's own series and almost always in conjunction with a peripheral defect. Hemianopia, both homonymous and heteronymous, was reported. The homonymous type was believed by Uhthoff to be due to complicating factors other than tabes. He expressed the belief that apparent heteronymous hemianopia is not a true hemianopic defect due to a chiasmal lesion but merely a symmetric defect of the second type, it having been noted by many authors that the changes in the fields in the two eyes are often the same, starting, for example, in both temporal fields or in both upper fields.

Rönne,[2] in his studies of the visual fields of a number of patients with tabetic atrophy of the optic nerve, found frequent nerve fiber bundle defects. He expressed the belief that the particular bundle of nerve fibers affected was more or less a matter of chance. Frequently a nasal bundle was involved and a temporal field defect produced. The papillomacular bundle might be affected and central scotoma produced, or occasionally there might be involvement of a paracentral bundle, producing an arcuate field defect somewhat similar to that seen in association with glaucoma. He noted that the scotoma associated with tabetic atrophy of the optic nerve was usually broader than that associated with glaucoma, and only seldom were other bundles entirely unaffected. He explained the infrequent involvement of the central field as probably due to the greater supply of nerve fibers to this region. Rönne noted a certain tendency toward symmetry of the fields in the two eyes but stated that this was not striking. He did not find homonymous defects in cases of tabetic atrophy of the optic nerve. The fields might by chance resemble those of hemianopia, but careful tests with test objects of graded sizes showed the defects not to be true hemianopia. He pointed out that true hemianopia is characterized by a sharp defect

2. Rönne, H.: Rührt die Optikusatrophie durch Tabes von einem Leiden der Ganglienzellen oder der Nervenfasern her? Arch. f. Ophth. **72**:481, 1909; Gesichtsfeldstudien über das Verhältnis zwischen der peripheren Sehschärfe und dem Farbensinn, speziell die Bedeutung derselben für die Prognose der Sehnervenatrophie, Klin. Monatsbl. f. Augenh. **49**:154, 1911; Ein Fall von Sehnervenatrophie bei Tabes mit einseitiger nasaler Hemianopsie, ibid. **50**:452, 1912.

across the vertical meridian and by the same limits in this region for all sizes of test object. He expressed the belief that the occurrence of nasal steps and the absence of hemianopic defects argue for a lesion starting somewhere in the optic nerve and not in the chiasm, the tracts or the ganglion cells of the retina.

Fuchs [3] reported some supposedly atypical field defects in cases of tabetic atrophy of the optic nerve. The total number of patients studied was not stated. He found no less than 30 cases of central scotoma and expressed the belief that the incidence of this condition is greater than the estimate of 2 per cent made by Uhthoff. Fuchs found central scotoma in cases of tabes to be nearly always bilateral. In the beginning it was central, but later it extended to the temporal side and became cecocentral. At first the scotoma was for color only, especially for red and for green. As it progressed, the peripheral field became contracted. Fuchs observed 6 cases of bitemporal hemianopia. He stated the belief that its bilateral occurrence could not be an accidental result of lesions located symmetrically in the two optic nerves, for he did not find binasal defects. He concluded, therefore, that in these 6 cases of bitemporal defect the lesion was located in the chiasm.

Langenbeck [4] studied the visual fields in 130 cases of tabetic atrophy of the optic nerve. In 89 of these the changes were characteristic of Uhthoff's first type. There were reduced acuity, partial or complete loss of color sensation and peripheral contraction, not necessarily concentric. In many cases the changes in the fields for color indicated the future course of changes in the field for form. In 27 cases the fields corresponded to Uhthoff's second type. They showed sector-shaped defects or (in 7 cases) marked concentric contraction, with well preserved function in the remaining part of the field. In 14 cases there was central scotoma, which was in no way like that seen in cases of retrobulbar neuritis. In 2 cases there was a defect somewhat similar to the nasal steps described by Rönne, but its boundary did not exactly follow the horizontal meridian. There were many instances of symmetric defects in the two eyes but no instances of true hemianopic defects.

Stargardt [5] reviewed the previous reports and emphasized the great diversity of defects in the fields in cases of tabetic atrophy of the optic nerve. He expressed the belief that, in addition to the various forms of peripheral contraction, two other types of defect, namely, central

3. Fuchs, E.: The Field of Vision in Tabetic Atrophy of the Optic Nerve, Tr. Am. Ophth. Soc. **12**:718, 1911.

4. Langenbeck, K.: Die Gesichtsfeldformen der tabischen Sehnervenatrophie, Klin. Monatsbl. f. Augenh. (pt. 2) **50**:148, 1912.

5. Stargardt: Ueber die Ursachen des Sehnervenschwundes bei der Tabes und der progressiven Paralyse, Arch. f. Psychiat. **51**:711, 1913.

scotoma and hemianopia, must be included. He stated that, although occasionally there were symmetric defects in the two eyes, in general this was not the case; he mentioned instances in which one eye was severely affected while the other was completely normal. He stated that he did not believe, therefore, that all the various reported cases of heteronymous hemianopia should be considered as instances of symmetric defects in the two eyes, or that homonymous defects could be attributed in all cases to a complicating cerebral or basal lesion.

Igersheimer [6] reported observations which indicated that changes in the fields may precede pallor of the disks. In a number of cases in which the older perimetric methods were used, he found questionable early defects associated with normal or nearly normal nerve heads. He was able to make prolonged observation in only 1 of these cases, in which at the first examination there were complete atrophy of the optic nerve and total blindness in the left eye. The right disk showed only a doubtful pallor, but there were marked defects in the peripheral field. The central visual acuity and the central color vision were entirely normal. One year later the patient reported by letter that he was totally blind in this eye. Modern perimetric methods proved more fruitful. When a tangent screen and small test objects were used in the examination, many tabetic patients with normal fundi showed nerve fiber bundle defects extending from the blindspot. A majority of these defects diminished or disappeared after antisyphilitic treatment. In 1 case, however, the defect disappeared before any treatment was given.

The more recent articles contribute little further to the knowledge of field defects in cases of atrophy of the optic nerve. Paton [7] reported perimetric studies in 8 cases to illustrate the variability in the defects. Arlt [8] examined a group of 53 patients and found that 85 per cent had contracted fields for form and color and reduction in acuity, 9 per cent had sector-shaped defects and 6 per cent had central scotomas. John,[9] in a study of the frequency of tabetic atrophy, gave incidental data on the frequency of central scotoma before and after the introduction of arsphenamine. In 1905 none of the 22 patients studied had central scotoma. In 1910 2 of 24 patients had central scotoma. In 1920 and in 1925, after the introduction of arsphenamine, central scotoma was found in 2 of 19 patients and in 4 of 18 patients, respectively. Thus, in the entire group of 83 patients there were 8, or about 10 per cent, with central scotoma.

6. Igersheimer, J.: Syphilis und Auge, Berlin, Julius Springer, 1918.

7. Paton, L.: Tabes and Optic Atrophy, Brit. J. Ophth. **6**:289, 1922.

8. Arlt, E.: Behandelte und unbehandelte Fälle von tabischen Sehnervenatrophie, Ztschr. f. ärztl. Fortbild. **19**:367, 1922.

9. John, I.: Ueber die Haüfigkeit der tabischen Optikusatrophie und ueber die bisherigen Behandlungsmethoden, Ztschr. f. Augenh. **69**:283, 1929.

Goldberg,[10] in an attempt to determine the initial field changes in cases of tabes, made perimetric studies of 75 tabetic patients, most of whom had no subjective symptoms of visual failure. Eight patients showed field changes, bitemporal in 7 cases. These were interpreted by Goldberg as due to lesions in the neighborhood of the chiasm.

Rutherford [11] reported studies of the visual fields of 50 patients with neurosyphilis of different types. Atrophy of the optic nerve was present in 7 of the 100 eyes and papilledema in 2. The other eyes presumably showed normal fundi, although this was not stated in the paper. The studies of the visual fields were made with a tangent screen at a distance of 1,200 mm. and a 1 mm. white test object (equivalent to approximately $\frac{1}{20}$ degree, or 3 minutes, in angular measure). The peripheral limits for this test object were recorded at intervals of 15 degrees around the circumference, and the blindspots were mapped. The author expressed the belief that the normal field for the 3 minute white test object should extend to 30 degrees in each of the four principal meridians. In 84 of the 100 eyes the limits of the field were less than this in one or more of the principal meridians. The blindspot was enlarged in 31 eyes. No statement was made as to the size of the blindspot considered normal for this test object. The central visual acuity ranged from 6/6 to 6/21—. There was no relation between the extent of the field and the degree of visual acuity. The author made no mention of central or of paracentral scotoma. The most marked defects occurred in the 7 eyes in which there was atrophy of the optic nerve.

PATHOLOGIC PICTURE OF PRIMARY ATROPHY OF THE OPTIC NERVE

The various pathologic studies of syphilitic atrophy of the optic nerve have given somewhat conflicting results. These have already been outlined in detail by Moore.[12] For our purposes they may be summarized as follows:

Stargardt [5] formed the following conclusions from extensive pathologic studies in 24 cases of atrophy of the optic nerve in association with dementia paralytica, the tabetic form of dementia paralytica and tabes. The lesion in the nerves consists in a primary peripheral and interstitial neuritis with secondary degeneration of the nerve fibers. The most frequent sites of the exudative process are the intracranial and the foraminal segment of the nerve, and next in frequency is the chiasm.

10. Goldberg, F.: Ueber initielle Veränderungen des Gesichtsfeldes bei Tabes im Zusammenhang mit der Frage der primären Lokalization der Läsion der Sehbahnen, Sovet. vestnik oftal **6**:508, 1935.

11. Rutherford, C. W.: Central Visual Fields and Pupils in Neurosyphilis, Tr. Am. Ophth. Soc. **33**:81, 1935.

12. Moore, J. E.: The Syphilitic Optic Atrophies with Especial Reference to Primary Optic Atrophy, Medicine **11**:263, 1932.

The orbital portion of the optic nerve, the tract and the lateral geniculate body are only rarely affected. In cases of paralysis the exudative process spreads from the brain to the optic pathways. In cases of tabes there is an isolated process in the optic nerve or the chiasm which may spread to the brain.

Wilbrand and Saenger [13] suggested that in cases in which there is a relatively early decrease in central acuity with only slight peripheral contraction the degenerative process might begin in the orbital part of the optic nerve, where the papillomacular bundle is peripherally located.

Igersheimer [14] summarized the results of his pathologic investigations as follows: Histologic evidence of atrophy in the nerve may occur in cases in which the ophthalmoscopic examination shows no abnormality. In the beginning the degenerative process is always at the periphery of the optic nerve. Inflammatory changes in the connective tissue do not necessarily occur. The findings in 2 cases (in 1 of which there were central scotoma and degeneration of the papillomacular bundle) show that the process can be distal or retrobulbar. The most frequently occurring form is diffuse. Another form, never before described, has as its characteristic sign a circumscribed focus.

Wagner von Jauregg [15] criticized the previous pathologic studies and expressed the belief that valid conclusions could be drawn only from a study of cases of pure tabes when atrophy of the optic nerve is in an early stage.

Although the majority of the pathologic studies support the view that the atrophic process starts in the marginal fibers of the intracranial portion of the optic nerve, distal to the chiasm, Stargardt's findings indicate that it may start at the chiasm; and Igersheimer's, that it may start in the orbital section of the optic nerve. As pointed out by Wagner von Jauregg, however, little is known of the pathologic changes characteristic of early uncomplicated tabes.

PRESENT STUDY

MATERIAL

The subjects for this study were 433 syphilitic patients. At the first examination 145 of these were discarded on account of some other complicating ocular disease, such as syphilitic choroiditis or glaucoma. There remained 288 patients for study. Of these 288 patients, 185 showed normal vision, visual fields and fundi on repeated examinations, and 103 showed evidences of involvement of the optic

13. Wilbrand, H., and Saenger, A.: Die Neurologie des Auges, Wiesbaden, J. F. Bergmann, 1913, vol. 5, chap. 22, p. 543.

14. Igersheimer, J.: Ueber der Opticusprozess bei Tabes und Paralyse, Deutsche med. Wchnschr. **52**:943, 1926.

15. Wagner von Jauregg, J.: Ueber tabische Optikus-Atrophie und deren Behandlung, Ztschr. f. Augenh. **61**:127, 1927.

neuro-pathways. Of the 103 patients with such involvement, 41 were examined only once and then disappeared from observation, while 62 were observed over periods varying from one month to seven years.

METHODS

The ophthalmologic studies consisted of examination of the external eyes, of the pupillary reactions and of extraocular movements; measurement of the tension; ophthalmoscopic examination; determination of the corrected visual acuity; tests of the light sense, and perimetric studies. The results of the tests of the light sense will be reported in a separate paper.

All the objective examinations of the eye were made by a single examiner (A. C. W.). The following terms are used in this report in clinical evaluation of the degree of change in the disks from normal to complete primary atrophy: normal disks; questionable pallor; early primary atrophy; moderate primary atrophy, and advanced primary atrophy. All the subjective tests, i. e., determinations of acuity, of light sense and of visual fields, were likewise made by one examiner (L. L. S.). The Ferree-Rand perimeter and tangent screen were used in charting the visual fields. In order to determine quantitatively the severity of the defects in different parts of the field, two or more test objects were used, selected from a graded series of white, blue and red test objects of 3, 1 and ½ degrees. The 1 and the ½ degree white test objects were particularly useful in detecting slight defects in the far periphery; and the 1 degree colored objects, in testing the central and the midperipheral region. The 3 degree white test object was used in those cases in which marked defects were found with the 1 and the ½ degree white object. The ½ degree blue and red test objects were used in detecting early defects in the central and the paracentral field in cases in which fields were normal for 1 degree white and colored objects.

Throughout this paper the region in which a given test object appeared weaker or dimmer than in the surrounding regions is designated as a relative scotoma for that particular test object. The term absolute scotoma is used to designate an area of complete blindness for a particular white or colored test object. For example, in a given region there might be relative scotoma for a ½ degree white and absolute scotoma for a 1 degree red object or relative scotoma for a 1 degree red and absolute scotoma for a ½ degree red object. Because of the greater reproductibility of the limits of an absolute scotoma, a test object sufficiently small to demonstrate an absolute scotoma was used whenever possible, rather than a larger object which could show only a relative scotoma.

RESULTS

The patients on whom these studies are based fell into four general clinical groups: (*a*) patients with primary atrophy of the optic nerve (56); (*b*) patients who had normal fundi but nevertheless showed defects in the visual fields similar to those found in patients with frank primary atrophy of the optic nerve (12); (*c*) patients with normal fundi or such lesions of the fundi as retinal arteriosclerosis or questionable pallor of the disks but with the common symptom of homonymous defects in the fields (12), and (*d*) patients with either active optic neuritis or a condition of the fundi indicative of a previous inflammatory process in the optic nerves (23).

The results of the clinical and perimetric studies on these different groups of patients are presented in four separate sections.

(*a*) *Patients with Clinically Diagnosed Syphilitic Primary Atrophy of the Optic Nerve.*—There were 56 patients with primary atrophy of the optic nerve among the 106 syphilitic patients with involvement of the optic neuro-pathways. The changes in the fields of these 56 patients were of four distinct types, as follows: (1) concentric peripheral contraction of the field (in 7); (2) localized defect in the peripheral field (in 19); (3) central or cecocentral scotoma with a normal peripheral field (in 8), and (4) central or cecocentral scotoma with a contracted peripheral field (in 22).

Type 1: The first type is characterized by an approximately concentric contraction of the field. In the early stages the defect is limited to the far periphery; the fields for colors and the central visual acuity are normal, while the fields for ½ degree and 1 degree white test objects show a moderate concentric contraction. The nerve head at this stage may show slight pallor or may look entirely normal. As the field defect progresses gradually inward, the fields for colors, particularly for red, show moderate concentric contraction. The nerve head at this stage shows slight or moderate pallor, but the central visual acuity remains normal. In the advanced stages the limits for a 1 degree white object show marked contraction; the field for a 1 degree red object may be reduced to a small central patch or be lost altogether; the visual acuity is moderately reduced, and the nerve head shows definite pallor.

Seven of the entire group of 56 patients with primary atrophy of the optic nerve showed this type of field defect. The findings for these 7 patients are summarized in table 1. To illustrate the various stages of this form of defect, selected perimetric charts for these 7 patients are given (figs. 1-3). The fields in cases 1, 2 and 3 (fig. 1) illustrate an early stage, which may be present before there is ophthalmoscopic evidence of atrophy of the optic nerve. In case 1, for example, at the time of the first perimetric test both disks appeared normal; four months later the right disk showed slight pallor but the left was still normal. In case 2 there was definite pallor of the right disk but only questionable pallor of the left. In case 3, at the time of the first perimetric tests the right disk showed questionable pallor and the left was normal. At the time of the second examination, six months later, there was moderate primary atrophy of the right disk; the left was still normal. The fields for the second date are shown in figure 1.

A somewhat more advanced stage of concentric contraction is illustrated in case 4. The fields (fig. 2) were plotted on a tangent screen of 30 degree radius and are shown on large scale charts covering only this portion of the field. With the right eye a ½ degree red test object

TABLE 1.—*Patients with Concentric Peripheral Contraction of Fields (Type 1)*

Case Number and Date Examined	Observations in Visual Fields	Appearance of Nerve Heads	Corrected Visual Acuity	Type of Syphilis	Progressive Changes in Visual Fields
1	.....	.....	.....	Tabes	Followed for only 3½ mo.; decrease in fields
11/27/34	O. U. Moderate concentric contraction	O. U. Normal	O. U. 20/15		
3/12/35	O. D. Increased contraction O. S. No change (fig. 1)	O. D. Early pallor O. S. Normal	O. U. 20/15		
2	O. U. Slight concentric contraction (fig. 1)	O. D. Early pallor O. S. Questionable pallor	O. U. 20/15	Diffuse meningovascular	Seen only once
3	.....	.....	.....	Diffuse meningovascular	Decrease in fields, followed by improvement
5/22/34	O. U. Moderate concentric contraction	O. D. Questionable pallor O. S. Normal	O. U. 20/15—1		
11/15/34	O. U. increased contraction (fig. 1)	O. D. Moderate primary atrophy of optic nerve O. S. Normal	O. U. 20/15—1		
11/6/36	O. U. Improvement	O. D. Moderate primary atrophy of optic nerve O. S. Pallor of peripheral rim of nerve tissue temporally and above	O. D. 20/15—3 O. S. 20/15		
4	O. U. Marked concentric contraction (fig. 2)	O. U. Advanced primary atrophy of optic nerve	O. D. 20/15 O. S. 20/30	Dementia paralytica	Seen only once
5	O. D. Blind O. S. Marked concentric contraction (fig. 3)	O. D. Advanced primary atrophy of optic nerve O. S. Moderate primary atrophy of optic nerve	O. D. Blind O. S. 20/30	Tabes	Seen only once but reported totally blind 4 yr. later
6	O. D. Perception of light only O. S. Marked concentric contraction (fig. 3)	O. U. Advanced primary atrophy of optic nerve	O. D. Perception of light only O. S. 20/70	Dementia paralytica	Seen only once
7	.....	.....	.....	Tabes	Decrease in fields between 1932 and 1936; questionable improvement in 1937
1/30/32	O. D. Blind O. S. Marked concentric contraction	O. U. Advanced primary atrophy of optic nerve	O. D. Blind O. S. 20/50+2		
1/25/35	O. S. Greater contraction	O. U. Same	O. S. 20/50—1		
2/14/36	O. S. Greater contraction (fig. 3)	O. U. Same	O. S. 20/50+2		
10/4/37	O. S. Questionable improvement	O. U. Same	O. S. 20/50		

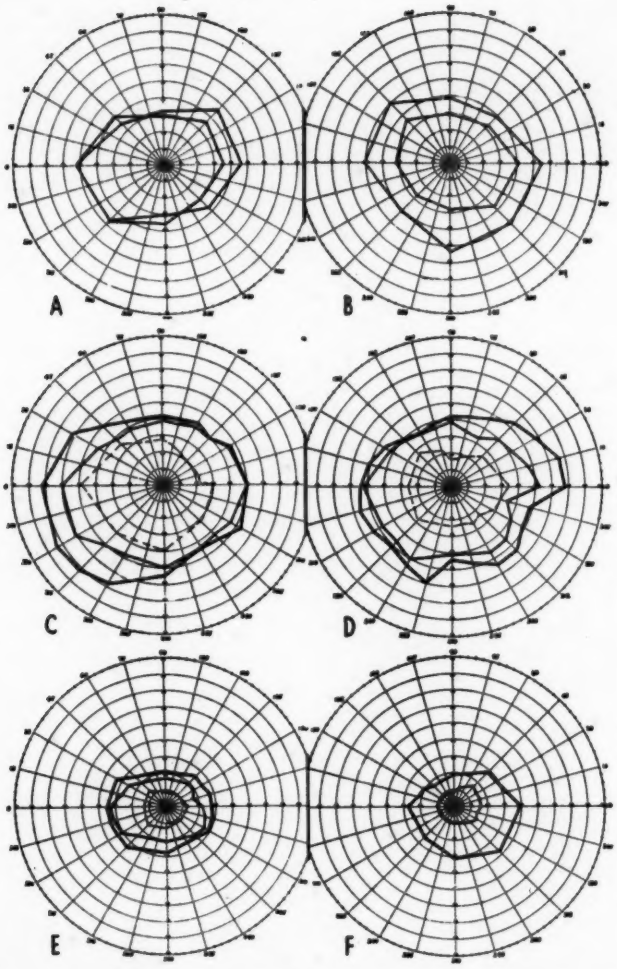

Fig. 1.—Early stage of concentric contraction (type 1). *A* and *B* (case 1) show the left and the right visual field, respectively, for a 1 degree white test object. The larger fields in *A* and *B* are those mapped on Nov. 27, 1934. The smaller fields, those mapped on March 12, 1935. The visual acuity of both eyes was 20/15 and the fundi were normal at the time of the first determination. At the second examination the right disk showed slight pallor. *C* and *D* (case 2) represent the left and the right visual field for 1 degree red (broken line), ½ degree white (inner solid line) and 1 degree white (outer solid line) test objects. There was questionable pallor of the left disk and only primary atrophy of the optic nerve. Visual acuity for both eyes was 20/15. *E* and *F* (case 3) represent the left and the right visual field for 1 degree red (broken line), 1 degree white (inner solid line) and 3 degree white (outer solid line) test objects on Nov. 15, 1934. The left eye showed a normal fundus, and moderate primary atrophy of the optic nerve was present in the right. Vision for both eyes was 20/15 — 1.

was seen in a small area extending from 2 to 4 degrees from the fixation point, showing that in spite of the marked contraction the foveal area was still relatively intact. In the left eye the limits of the field for a ½ degree red object, though wider, approached the fixation point in the upper and the temporal meridian. The visual acuity was 20/15 in the right eye and 20/30 in the left. Both disks showed advanced primary atrophy of the optic nerve.

In cases 5, 6 and 7 the vision in one eye was reduced to light perception or to total blindness. In the better eye there was moderate reduction of visual acuity, to 20/30, 20/70 and 20/50, respectively. The disks showed advanced atrophy, and the fields for a 1 degree white test object were markedly contracted in every case. In cases 5 and 7, a 1 degree red object was seen in a small central area. In case 6, a 3 degree red test object was not seen anywhere as red. The patient, it should be noted, had a lower visual acuity than did the 2 who still

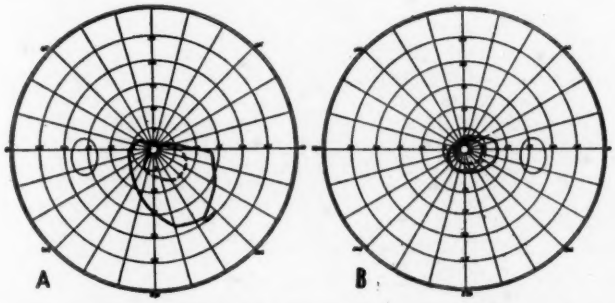

Fig. 2 (case 4).—Visual field of (*A*) left and (*B*) right eye, showing advanced stage of concentric contraction, with preservation of good central vision (type 1) in a case of advanced primary atrophy of the optic nerve. The broken line bounds the field for a ½ degree red and the solid line for a 1 degree white test object. Visual acuity for the left eye was 20/30 and for the right 20/15.

retained some perception of color at the fovea. In case 7 the fields at three different times are given, to show the gradual decrease during four years (fig. 3). During this time the visual acuity remained unchanged.

On account of the late involvement of the fovea with this type of field defect, the central visual acuity gives little or no indication of the degree of involvement of the optic nerve. The findings in cases 1 and 3 indicated that fairly marked field defects can be present before there is ophthalmoscopic evidence of atrophy of the optic nerve. When first observed, the disks in case 1 were classed as entirely normal, but there was concentric contraction of the visual fields. Three months later the right eye showed definite pallor, and the visual field was further reduced. In case 3 there was questionable pallor of the right disk and

a normal left disk at the time of the first examination; the visual fields were contracted. Six months later the atrophy was definite in the right eye, and the visual field was further reduced. Two years later there was improvement in the fields of both eyes, but the nerve heads were essentially unchanged.

Type 2: A second and more common type of field defect is that characterized not by concentric contraction but by a defect particularly marked in some one section of the field. Table 2 summarizes the find-

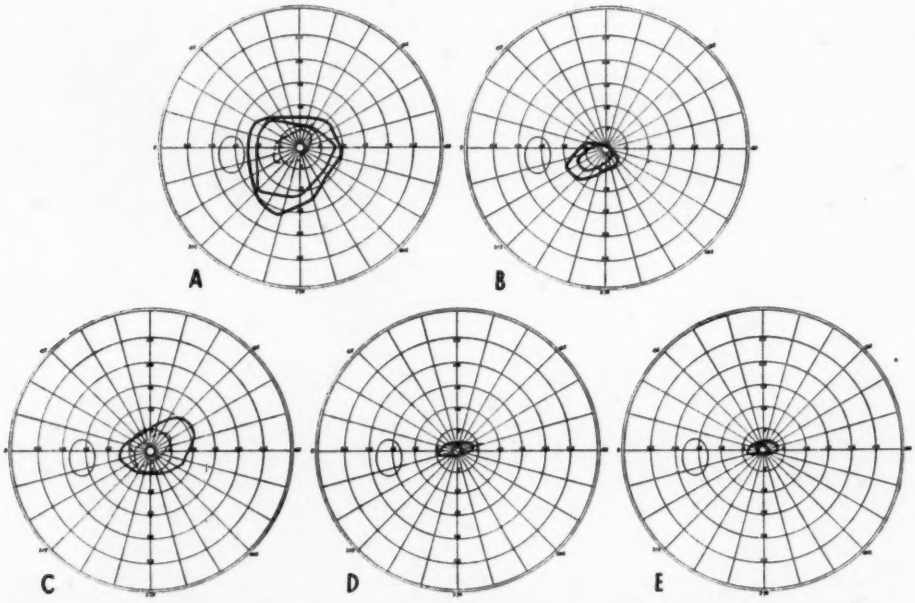

Fig. 3.—Advanced stage of concentric contraction, associated with reduced central vision (type 1). *A* (case 5) shows the left visual field for 1 degree red (broken line), 1 degree white (inner solid line) and 3 degree white (outer solid line) test objects in a case of moderate primary atrophy of the optic nerve. Vision was 20/30. *B* (case 6) shows the left visual field for 1 degree white (inner line) and 3 degree white (outer line) test objects in a case of advanced primary atrophy of the optic nerve. Vision was 20/70. *C, D* and *E* (case 7) show the left visual field in a case of advanced primary atrophy of the optic nerve as of Jan. 30, 1932 (*C*), Jan. 25, 1935 (*D*), and Feb. 14, 1936 (*E*). The solid line bounds the field for a 1 degree white test object and the broken line that for a 1 degree red test object. Vision at the first and the third determination was 20/50 +2 and at the second determination 20/50 —1.

ings for the 19 patients whose fields were of this type.[16] As with type 1, the central visual acuity may be normal in the early stages when

16. In 10 of these, as has been indicated in the table, the classification as to type of defect in field was somewhat uncertain because of poor cooperation and unsteady fixation or because of advanced defects in field.

the foveal region is not involved, and there may likewise be only slight pallor of the disk. In general, however, reduction of acuity occurs at an earlier stage than with type 1, because the localized field defect progresses inward and involves the central region sooner than does a defect characterized by concentric contraction.

Selected charts illustrating various stages and forms of this type of defect are shown in figures 4 and 5. The fields in 4 cases (8, 9, 10 and 11) in which there were early field changes and normal visual acuity are shown in figure 4. In case 8 the right eye had a normal disk and a normal visual field. The left eye showed early atrophy of the optic nerve. The lower half of the visual field showed contraction for both white and red test objects, while the upper half of the field was entirely normal. Under treatment this patient ultimately showed complete restoration of the visual field. In case 9 the right eye showed only a slight cut in the lower nasal field and the left eye a more advanced defect limited chiefly to the nasal field. There was bilateral early atrophy of the optic nerve. In case 10 both eyes showed almost total loss of the upper portion of the field for a 1 degree white test object and moderate contraction in the lower portion. The disks showed moderate pallor. In case 11 the right eye was blind. The field defect in the left eye was limited entirely to the upper temporal quadrant. In this quadrant the limits for a 1 degree red test object came close to but did not involve the fixation point. Visual acuity was consequently not reduced. Examination of the fundi showed advanced atrophy of the optic nerve on the right and early atrophy on the left.

The fields in cases 13, 14, 15 and 16, shown in figure 5 for one eye only, illustrate a more advanced stage. The localized defect extended from the periphery and involved the foveal region, with a resultant reduction in acuity. In case 13 the defect for a 1 degree white test object in the upper field extended to the fixation point, and neither the 1 degree blue nor the 1 degree red test object was seen in this region. The visual acuity was 20/100. In case 14 the defect for the 1 degree white and for the 1 degree blue object were limited almost entirely to the nasal field. A 3 degree red test object, however, was not seen anywhere as red. The vision was 10/200. There was bilateral advanced atrophy of the optic nerve. The fields in case 15 were somewhat similar. Complete loss of vision for all colors, however, indicated a greater involvement of the temporal field than was present in case 14. In case 16 the fields showed a partial recovery of function in the foveal region a year after the first determination. In May 1933 the 1 degree blue test object was seen only in a small paracentral area of the temporal field. The visual acuity was 20/70 + 2. In April 1934 there was a slight increase in the field for the blue test object, which was seen at

TABLE 2.—*Patients with Localized Defects in Peripheral Fields (Type 2)*

Case No. and Date Examined	Observations in Visual Fields	Appearance of Nerve Heads	Corrected Visual Acuity	Type of Syphilis	Progressive Changes in Visual Fields	Comment
8	.....	.....	.....	Incipient dementia paralytica	Complete recovery	
3/19/31	O. D. Normal O. S. Defect in lower field	O. D. Normal O. S. Early primary atrophy of optic nerve	O. U. 20/15			
12/21/31	O. D. Normal O. S. Improvement	O. U. Same	O. U. 20/15			
1/18/35	O. U. Normal (fig. 4)	O. U. Same	O. U. 20/15			
9	O. D. Slight contraction in lower nasal field O. S. Contraction, especially of nasal field (fig. 4)	O. U. Early primary atrophy of optic nerve	O. U. 20/15	Dementia paralytica	No change when retested 7 mo. later	
10	O. U. Contraction, chiefly of upper field (fig. 4)	O. U. Moderate primary atrophy of optic nerve	O. U. 20/15	Tabes	Seen only once	
11	O. D. Blind O. S. Contraction in upper temporal quadrant (fig. 4)	O. D. Advanced primary atrophy of optic nerve O. S. Early primary atrophy of optic nerve	O. D. Blind O. S. 20/20	Tabes	Seen only once	
12	O. D. Advanced contraction O. S. Contraction, especially of temporal field	O. U. Moderate primary atrophy of optic nerve	O. D. 20/200 O. S. 20/30	Diffuse meningo-vascular	Followed for 11 mo. only; successive tests showed decrease in fields	
13	O. D. Normal O. S. Contraction, especially of upper field (fig. 5)	O. D. Normal O. S. Advanced primary atrophy of optic nerve	O. D. 20/15 O. S. 20/100	Tabes	Followed for 2 yr.; no change	
14	O. D. Contraction, especially of nasal field O. S. Blind (fig. 5)	O. U. Advanced primary atrophy of optic nerve	O. D. 5/100 O. S. Blind	Diffuse meningo-vascular	Seen only once	
15	O. D. Contraction, especially of nasal field O. S. Light perception in temporal field only (fig. 5)	O. U. Moderate primary atrophy of optic nerve	O. D. 14/200 O. S. Light perception	Dementia paralytica	Seen only once	
16	.....	.....	.....	Dementia paralytica	Improvement	
5/ 1/33	O. D. Contraction, especially in nasal field O. S. Questionable light perception in lower temporal field	O. U. Advanced primary atrophy of optic nerve	O. D. 20/70+2 O. S. Questionable light perception			
4/20/34	O. D. Improvement in field for blue O. S. No change	O. U. Advanced primary atrophy of optic nerve	O. D. 20/40—1 O. S. Questionable light perception			

17	O. D. Advanced contraction O. S. Questionable light perception	O. U. Advanced primary atrophy of optic nerve	O. D. 20/100 O. S. Questionable light perception	Diffuse meningovascular	Became blind	Advanced defect in field, difficult to classify as to type
18	O. D. Blind O. S. Advanced contraction, mostly temporal	O. U. Advanced primary atrophy of optic nerve	O. D. Blind O. S. 12/200	Diffuse meningovascular	Followed for 5½ yr.; successive tests showed decrease in fields	Advanced defect in field, difficult to classify as to type
19	O. D. Advanced contraction, mostly nasal O. S. Questionable light perception	O. U. Advanced primary atrophy of optic nerve	O. D. 6/200 O. S. Questionable light perception	Dementia paralytica	Became blind	Advanced defect in field, difficult to classify as to type
20	O. D. Blind O. S. Advanced contraction in nasal and in upper field	O. U. Advanced primary atrophy of optic nerve	O. D. Blind O. S. 4/200	Tabes	Followed for only 4½ mo.; fields became worse	Advanced defect in field, difficult to classify as to type
21	O. D. Questionable light perception O. S. Advanced contraction, mostly of temporal field	O. U. Advanced primary atrophy of optic nerve	O. D. Questionable light perception O. S. 5/200	Congenital	Seen only once	
22	O. U. Advanced contraction, greatest in lower field	O. U. Advanced primary atrophy of optic nerve	O. D. 20/70—2 O. S. 20/50—1	Tabes	Followed for 1½ yr.; no change	Fields suggest altitudinal hemianopia; similar to those in case 10 except that lower instead of upper fields are involved
23	O. D. Advanced contraction, greatest in lower and lower temporal field O. S. Blind	O. U. Advanced primary atrophy of optic nerve	O. D. 10/200 O. S. Blind	Diffuse meningovascular	Followed for 2 yr.; no change	Fields difficult to classify as to type because of advanced defect
24	O. D. Advanced contraction, particularly in upper field O. S. Same as O. D.	O. U. Moderate pallor and waxy appearance of nerve heads	O. D. 20/100 O. S. 20/200	Clinical syphilis of central nervous system; unclassified (no spinal puncture)	Seen only once	Fields difficult to classify as to type because of advanced defect
25	O. D. Moderate contraction, mostly of temporal field O. S. Same as O. D.	O. U. Early primary atrophy of optic nerve	O. D. 20/40+2 O. S. 20/30—2	Tabes	Followed for only 8 mo.; no change	Perimetric data unreliable because of unsteady fixation and poor cooperation
26	O. D. Light perception in upper nasal field only O. S. Marked contraction, leaving only upper nasal field for 3° white	O. U. Advanced primary atrophy of optic nerve	O. D. Light perception O. S. Counting fingers at 6 in. (15 cm.)	Diffuse meningovascular	Followed for 6 yr.; between 4/21/31 and 5/5/36 gradual decrease; questionable improvement on 3/18/37	Difficult to classify as to type because of advanced defect; might be type 4

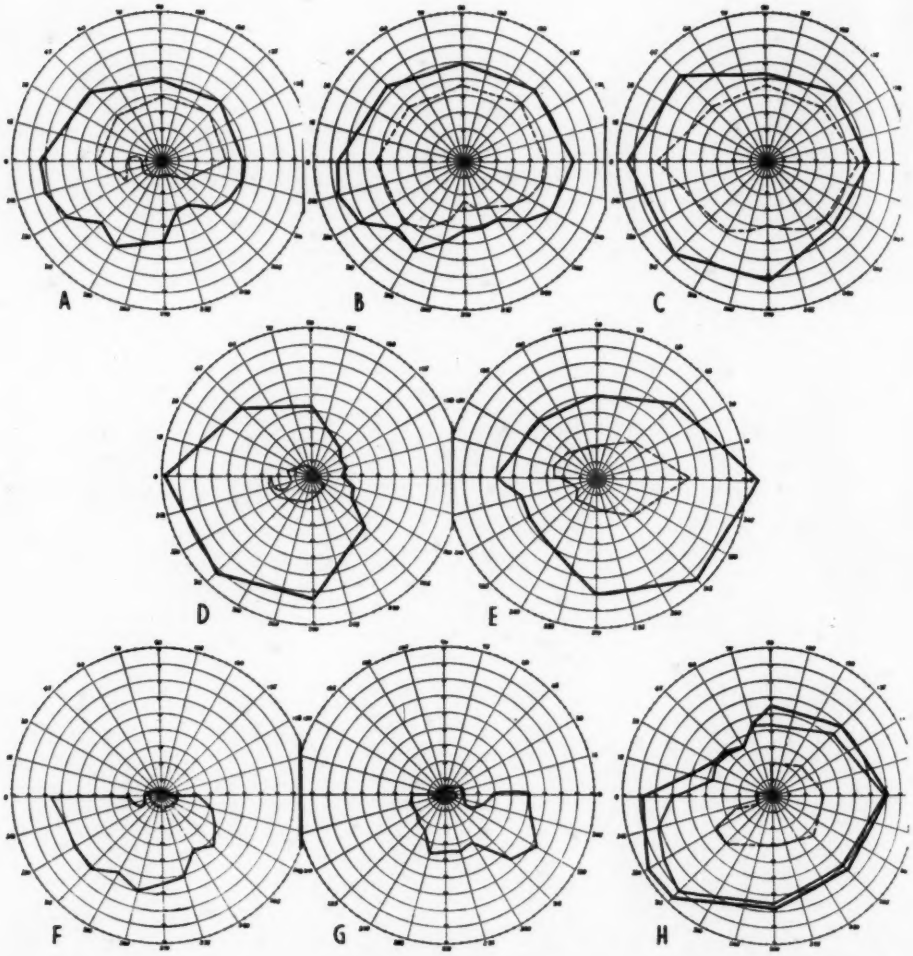

Fig. 4.—Various stages of localized peripheral defects, associated with normal central vision (type 2). *A, B* and *C* (case 8) show the left visual field in a case of early primary atrophy of the optic nerve on March 19, 1931, Dec. 21, 1931, and Jan. 18, 1935, respectively, for 1 degree white (solid line) and 1 degree red (broken line) test objects. Vision remained 20/15 throughout the period of observation. *D* and *E* (case 9) show the left and the right visual field, respectively, in a case of early primary atrophy of the optic nerve, the solid line bounding the field for a 1 degree white test object and the broken line that for a 1 degree red test object. Vision in both eyes was 20/15. *F* and *G* (case 10) show the left and the right visual field in a case of moderate primary atrophy of the optic nerve, the solid line bounding the field for a 1 degree white test object and the broken line, that for a ½ degree red test object. Vision for both eyes was 20/15. *H* (case 11) shows the left visual field in a case of early primary atrophy of the optic nerve for 1 degree red (broken line), ½ degree white (inner solid line) and 1 degree white (outer solid line) test objects. Vision was 20/20.

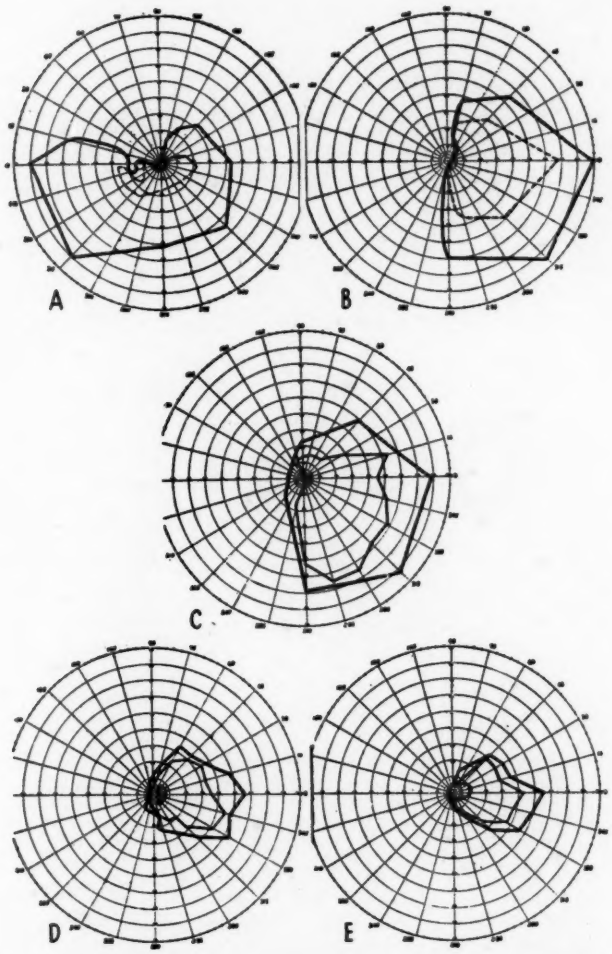

Fig. 5.—Localized defects, associated with reduced central vision (type 2). *A* (case 13) shows the left visual field in a case of advanced primary atrophy of the optic nerve, the solid line bounding the field for a 1 degree white test object and the broken line that for 1 degree red and blue objects. Vision was 20/100. *B* (case 14) shows the right visual field in a case of advanced primary atrophy of the optic nerve, the solid line bounding the field for a 1 degree white and the broken line that for a 1 degree blue test object. A 3 degree red test object was not seen. Vision was 5/100. *C* (case 15) shows the right visual field in a case of moderate primary atrophy of the optic nerve for a 3 degree white (outer line) and a 1 degree white (inner line) test object. A 3 degree blue and a 3 degree red test object were not seen. Vision was 14/200. *D* and *E* (case 16) show the right visual field in a case of advanced primary atrophy of the optic nerve on May 1, 1933, and April 20, 1934, respectively. The outer solid line bounds the field for a 1 degree white, the inner solid line that for a ½ degree white and the broken line that for a 1 degree blue test object. A 3 degree red test object was not seen on either occasion. Vision at the first determination was 20/70 + 2 and at the second 20/40 — 1.

that time in the central region, and there was a corresponding improvement in visual acuity, to 20/40 — 1.

As indicated in the illustrations and in table 2, the most marked defect may occur in any part of the field. It should be noted, however, that almost invariably the same region is affected in the two eyes. This tendency is illustrated in figure 4 (cases 9 and 10) and is evident from a study of the table.

Type 3: The third type of field defect differs markedly from the two previously considered. Central or cecocentral scotoma is associated with normal or nearly normal peripheral limits for a 1 degree white test object. Eight of the 56 patients had this type of defect. In a majority of these patients the disks showed atrophy of the papillomacular bundle or of the temporal portion. In 2 cases, however, although the perimetric studies indicated that the lesion involved chiefly the papillomacular bundle, localized temporal atrophy of the nerve was not observed on ophthalmoscopic examination. The visual acuity in cases of this type is involved early.

The fields in 3 cases (27, 28 and 29) illustrating this type of defect are shown in figure 6. In case 27 bilateral cecocentral scotomas were found with the 1 degree and the ½ degree white test object. Only the smaller of these test objects showed involvement of the foveal area. The visual acuity was 12/200 in the right and 20/200 in the left eye. The peripheral fields of both eyes were normal for a 1 degree white test object. Attempts to map the fields for 1 degree blue and red objects gave variable and inconsistent results but indicated a probable slight involvement of the peripheral field. Both disks showed atrophy of the papillomacular bundle. In case 28 there was a unilateral defect only, consisting of a paracentral scotoma for a 1/6 degree white test object and a larger cecocentral scotoma for a 1 degree red test object. No scotoma was demonstrable with a ½ degree white test object. The visual acuity was 20/70—. The peripheral fields for 1 degree white, blue and red test objects were entirely normal, indicating that the lesion was confined to the papillomacular bundle. The disk, however, showed moderate generalized atrophy. In case 29 the perimetric findings showed early involvement of the right eye and a more advanced defect in the left. Results for two different dates are shown in the illustration. On the first date the left eye showed a cecocentral scotoma for ½ degree white and 1 degree red objects. The scotoma for the white test object did not extend to the fovea. The left eye showed only a relative cecocentral scotoma for a ½ degree red object, but this did not involve the foveal region. The visual acuity was 20/15 —1 in the right eye and 20/20 —1 in the left. Both nerve heads showed early atrophy of the papillomacular bundle. The results of the perimetric examination made

TABLE 3.—*Patients with Central or Cecocentral Scotomas and Normal Peripheral Fields*

Case No. and Date Examined	Observations in Visual Fields	Appearance of Nerve Heads	Corrected Visual Acuity	Type of Syphilis	Progressive Changes in Visual Fields	Comment
27	O. U. Cecocentral scotoma (fig. 6)	O. U. Moderate atrophy of papillomacular bundle	O. D. 12/200 O. S. 20/200	Diffuse meningo-vascular	Seen only once	
28	O. D. Cecocentral scotoma O. S. Normal (fig. 6)	O. D. Moderate primary atrophy of optic nerve O. S. Normal	O. D. 20/70— O. S. 20/15	Diffuse meningo-vascular	Seen only once	
29 12/3/34	O. D. Cecocentral scotoma; fovea not involved O. S. Cecocentral scotoma involving fovea	O. U. Early atrophy of papillomacular bundle	O. D. 20/15—1 O. S. 20/20—1	Diffuse meningo-vascular	Scotoma became worse between 12/3/34 and 5/28/36; no change between 5/28/36 and 4/5/37	
5/28/36	O. U. Scotoma worse (fig. 6)	O. U. Advanced atrophy, especially of papillomacular bundle	O. D. 20/15—3 O. S. 20/40+3			
30	O. U. Central scotoma for 1° red	O. U. Questionable atrophy of papillomacular bundle	O. U. 20/20—3 (uncorrected)	Tabes	Seen only once	
31	O. D. Blind (glaucoma) O. S. Central scotoma for ½° white	O. D. Advanced glaucomatous atrophy O. S. Early primary atrophy of optic nerve	O. D. Blind (glaucoma) O. S. 15/200	Tabes	Followed for 4½ yr.; no change	Poor cooperation; unsteady fixation
32	O. U. Cecocentral scotomas for 3° and 1° white	O. U. Moderate atrophy of papillomacular bundle	O. D. 9/200 O. S. 6/200	Tabes	Followed for 8 mo. only; no change	
33	O. D. Cecocentral scotoma for 1° white O. S. Cecocentral scotoma for ½° white	O. U. Advanced atrophy, possibly more marked in area of papillomacular bundle	O. D. 5/200 O. S. 10/200	Tabes	Followed for 5 yr.; no change	
34	O. U. Relative cecocentral scotomas for ½° blue and ¼° white (red not used because of questionable congenital red-green color blindness)	O. U. Early pallor, especially of papillomacular bundle	O. D. 20/40 O. S. 20/50 (opacities in lens)	Congenital	Followed for 5½ yr.; no significant change	Reduced vision, due in part to deposits of pigment on anterior lens capsules; probable congenital red-green color blindness

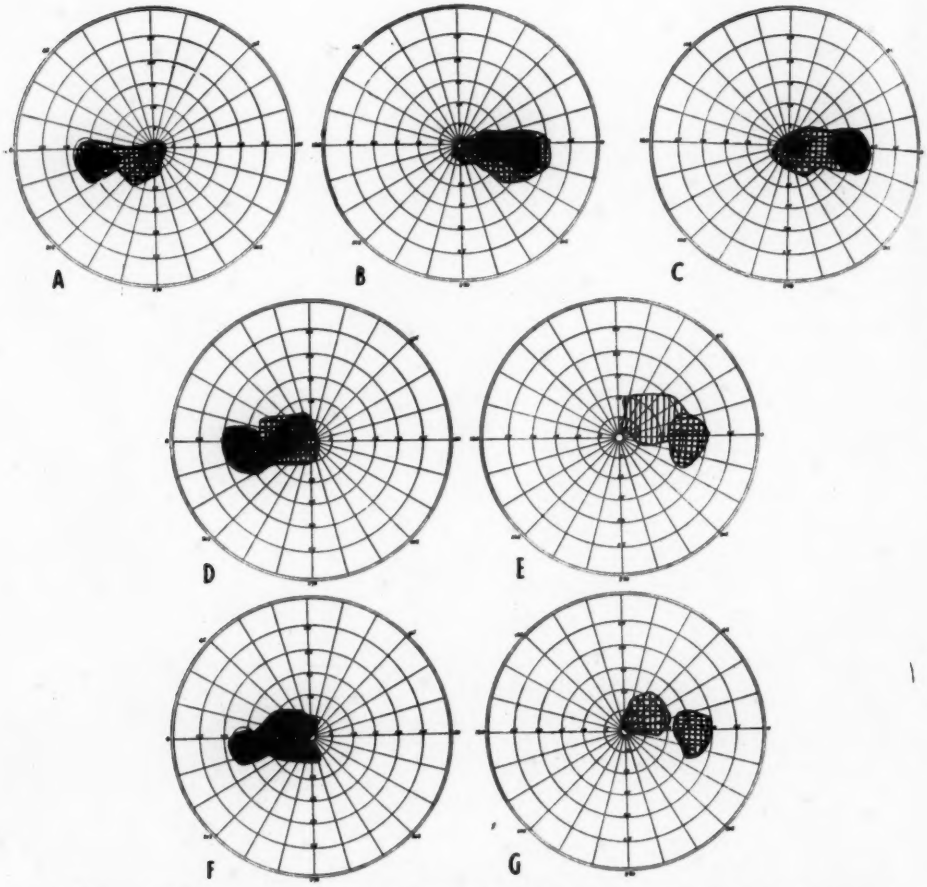

Fig. 6.—Cecocentral scotomas associated with normal peripheral fields (type 3). The central fields (to 30 degrees only) are shown in the figure. *A* and *B* (case 27) show a scotoma for 1 degree (solid area) and ½ degree (cross-hatched area) white test objects in the left and the right eye, respectively, in a case of moderate atrophy of the papillomacular bundle. Both peripheral fields for a 1 degree white test object were normal. Vision for the left eye was 20/200 and for the right 12/200. *C* (case 28) shows a scotoma in the right visual field for a ⅛ degree white (solid area) and a 1 degree red (cross-hatched area) test object in a case of moderate primary atrophy of the optic nerve. Vision was 20/70. *D*, *E*, *F* and *G* (case 29) show scotoma in the left and the right visual field on Dec. 3, 1934 (*D* and *E*) and May 28, 1936 (*F* and *G*). At the time of the first observation early atrophy of the papillomacular bundle was present and vision for the left eye was 20/20 — 1 and for the right eye 20/15 — 1. At the time of the second observation advanced atrophy, especially of the papillomacular bundle, was present and vision was 20/40 + 3 for the left eye and 20/15 — 3 for the right. The solid area in both charts of the left visual field represents scotoma for a ½ degree white test object. The cross-hatched areas in *D* and in *G* represent scotomas for a 1 degree red object. In *E* the area covered with vertical lines represents relative and the cross-hatched area absolute scotoma for a ½ degree red test object.

eighteen months later showed a slight increase in the density of the scotomas in both eyes. The right eye showed an absolute scotoma for a 1 degree red object extending up to but not covering the fixation point. The vision was 20/15 — 3. In the left eye the scotoma for a ½ degree white object had extended over the fixation point, and the visual acuity had decreased to 20/40 + 3. The ophthalmoscopic examination showed well advanced atrophy, particularly of the papillomacular bundles, and of about the same degree in both eyes.

Type 4: The defect of the fourth type is characterized by central or cecocentral scotoma, as in type 3, combined with a peripheral defect of either type 1 or type 2. There were 22 patients with this type of involvement. The findings are summarized in table 4. In a few cases advanced changes involving the central field were found at the first examination. It is difficult to say definitely whether these should be classified in type 2 or type 4. Such cases are indicated in the table.

In a majority of the patients with changes of this type, both the central and the peripheral defects were present at the first examination. These patients, like those with changes of type 3, showed an early reduction in visual acuity. Repeated studies of the field in 3 cases showed that the central scotoma developed later than the peripheral contraction. The especial involvement of the papillomacular bundle is sometimes apparent at ophthalmoscopic examination, but the most frequent finding is generalized atrophy similar to that seen with types 1 and 2.

Figures 7 to 12 illustrate defects of this type. In case 35 the fields of both eyes for the 1 degree white test object showed rather marked concentric contraction. The left eye showed in addition an absolute cecocentral scotoma for the 1 degree white object. The defects in the field of the right eye were less advanced. Both the 1 and the ½ degree white test object were seen in the central region, but there was a relative central scotoma for the smaller object. The peripheral limits for the 1 degree white object showed less contraction than did those of the left eye; 1 degree red and blue test objects were not recognized anywhere with either eye. The visual acuity was 10/200 for the right and 2/200 for the left eye. Both disks showed primary atrophy, especially marked in the papillomacular area.

In cases 36 and 37 central scotoma was combined with peripheral defects which were more marked in one region of the field. In case 36 the peripheral defect in the right eye was limited almost entirely to the upper temporal quadrant. In the left eye the defect was more advanced. The field for a 1 degree white test object showed a marked defect in the upper temporal region and a slight contraction in the remaining portions. No tests with colors were made of the left eye. The visual acuity was 20/200 in the right and 2/200 in the left eye. The disks

TABLE 4.—*Patients with Central Scotomas and Peripheral Defects (Type 4)*

Case No. and Date Examined	Observations in Visual Fields	Appearance of Nerve Heads	Corrected Visual Acuity	Type of Syphilis	Progressive Changes in Visual Fields	Comment
35	O. U. Concentric contraction and cecocentral scotoma (fig. 7)	O. U. Advanced atrophy, especially of papillomacular bundle	O. D. 10/200 O. S. 2/200	Diffuse meningovascular	Seen only once	
36	O. U. Cecocentral scotoma and defect in upper temporal field (fig. 8)	O. U. Advanced atrophy of papillomacular bundle	O. D. 20/200 O. S. 2/200	Dementia paralytica	Followed for 8 mo. only; slight improvement	
37	O. D. Large cecocentral scotoma and marked peripheral contraction O. S. Cecocentral scotoma and peripheral defect, especially in upper field (fig. 9)	O. D. Advanced atrophy, especially of papillomacular bundle O. S. Moderate atrophy, especially of papillomacular bundle	O. D. Hand motion O. S. 20/200+1	Diffuse meningovascular	Seen only once	
38	O. D. Cecocentral scotoma for 1° colors (fig. 10) O. S. 3° white seen only in nasal field	O. U. Advanced primary atrophy of optic nerve	O. D. 20/200 O. S. Light perception	Tabes	Followed for only 4 mo.; no significant change	
39	O. D. Cecocentral scotoma and marked peripheral contraction, especially in nasal field (fig. 10) O. S. Light perception only in lower field (questionable)	O. U. Advanced primary atrophy of optic nerve	O. D. 7/200 O. S. Hand motion	Tabes	Followed for 2 yr.; no significant change	
40	.....	.....	.....	Dementia paralytica	Followed for 2 yr.; fields became worse	
11/2/32	O. D. Cecocentral scotoma and peripheral defect, especially in temporal field O. S. 3° white seen in small area in nasal field	O. U. Advanced primary atrophy of optic nerve	O. D. 20/200 O. S. Finger counting at 6 in. (15 cm.)			
9/12/34	O. D. More advanced O. S. Light perception only in nasal field (fig. 11)	O. U. Advanced primary atrophy of optic nerve	O. D. 8/200 O. S. Light perception			
41	.....	.....	.....	Tabes	Followed for 2½ yr.; fields became gradually worse	
12/20/35	O. U. Central scotoma for 1° red; marked peripheral contraction for 1° white	O. U. Advanced atrophy, especially of papillomacular bundle	O. D. 20/100 O. S. 20/70—1			
6/19/36	O. U. Fields worse	.....	O. D. 20/200 O. S. 20/200+1			
1/12/37	O. U. Fields worse	.....	O. D. 10/200 O. S. 14/200			
4/28/37	O. U. Fields worse	.....	O. D. 4/200 O. S. 8/200			
11/2/37	O. U. Fields worse (fig. 12)	O. U. Advanced atrophy, especially of papillomacular bundle	O. D. 4/200 O. S. 5/200			
42	O. U. Central scotoma and slight peripheral contraction for 1° white	O. U. Moderate atrophy, especially of papillomacular bundle	O. D. 15/200 O. S. 15/200	Tabes	Seen only once	Poor cooperation

43	O. D. Absolute central scotoma for 1° blue; slight concentric contraction for 1° white O. S. Central scotoma, relative for 1° blue, absolute for ½° blue; slight concentric contraction for 1° white (red not used because of congenital red-green color blindness)	O. U. Advanced primary atrophy of optic nerve	O. D. 20/50 O. S. 20/40	Diffuse meningovascular	Followed for 6 yr.; no significant change	Congenital red-green color blindness
44	O. D. Normal O. S. Cecocentral scotoma for color and contraction of temporal field	O. D. Normal O. S. Moderate primary atrophy of optic nerve	O. D. 20/15—2 O. S. 20/70—20/100	Tabes	Followed for 5½ yr.; defect in peripheral field showed questionable improvement; no change in central scotoma	
45	O. D. Cecocentral scotoma and peripheral contraction in nasal field O. S. Marked contraction, mostly in upper field	O. U. Moderate primary atrophy of optic nerve	O. D. 20/200—12/200 O. S. 6/200	Diffuse meningovascular	Followed for 5½ yr.; no significant change	Perimetric data for O. S. unreliable because of unsteady fixation
46	O. U. Moderate peripheral contraction for 1° white; 1° blue and red not seen; 3° blue seen in midperiphery but not in central field	O. U. Moderate primary atrophy of optic nerve	O. D. 20/200+1 O. S. 20/200+1	Diffuse meningovascular	Followed for only 3 mo.; no change	Poor cooperation; classification as to type of field is doubtful
47	O. D. 3° white, 1° blue and 3° red seen only in lower nasal quadrant; 1° red not seen O. S. Light perception only	O. U. Advanced primary atrophy of optic nerve	O. D. 2/200 O. S. Light perception	Diffuse meningovascular	Followed for only 1 mo.; no change	Advanced defect in field; classification as to type uncertain
48	.....	.....	.....	Diffuse meningovascular	Followed for 16 mo.; gradual decrease	
6/28/33	O. U. Defects in cecocentral and in lower nasal field of nerve fiber bundle type	O. U. Moderate primary atrophy of optic nerve	O. D. 20/50—1 O. S. 20/50—1			
10/31/33	O. U. More advanced	O. U. Advanced primary atrophy of optic nerve	O. D. 20/200 O. S. 20/200			
2/24/36	O. U. More advanced (fig. 13)	O. U. Advanced primary atrophy of optic nerve	O. D. 4/200 O. S. 4/200			
49	.....	.....	.....	Diffuse meningovascular	Followed for 1½ yr.; slight decrease	
2/21/35	O. D. Scotoma extending from blindspot, involving fovea and lower nasal field (nerve fiber bundle type) O. S. Complete loss of all but upper nasal field	O. U. Moderate primary atrophy of optic nerve, especially of papillomacular bundle	O. D. 10/200 O. S. 6/200			
9/ 9/36	O. U. Slightly worse (fig. 14)	O. U. Advanced primary atrophy of optic nerve	O. D. 16/200 O. S. 6/200			
50	.....	.....	.....	Diffuse meningovascular	Followed for only 1 mo.; improvement	
3/8/37	O. U. Cecocentral scotoma and defect in lower nasal field (nerve fiber bundle type)	O. U. Moderate primary atrophy of optic nerve, more marked in O. D.	O. D. 3/200 O. S. 5/200			
4/2/37	Improvement (fig. 15)	O. U. No change	O. D. 5/200 O. S. 7/200			
51	O. D. Light perception in upper nasal field only O. S. Defects in cecocentral and in lower nasal field (similar to fields in cases 48, 49 and 50)	O. U. Advanced primary atrophy of optic nerve	O. D. Light perception O. S. 6/200	Diffuse meningovascular	Seen only once	

TABLE 4.—*Patients with Central Scotomas and Peripheral Defects (Type 4)—Continued*

Case No. and Date Examined	Observations in Visual Fields	Appearance of Nerve Heads	Corrected Visual Acuity	Type of Syphilis	Progressive Changes in Visual Fields	Comment
52	.....	.....	.....	Clinical of central nervous system, unclassified; seronegative cerebrospinal fluid	Followed for 1 yr. 7 mo.; no change during first year; decrease during second year	
1/19/35	O. D. Defect in lower nasal field for 1° white, extending from periphery to blindspot O. S. Defect in lower nasal field for 1° white	O. D. Early primary atrophy of optic nerve O. S. Normal				
1/10/36	O. U. No change	O. U. Early primary atrophy of optic nerve, more marked in O. D.	O. D. 20/30—3 O. S. 20/15			
9/ 8/37	O. U. Fields worse; defect in left field for 1° red now extends from lower nasal periphery to blindspot (nerve fiber bundle defects similar to those in cases 48 to 51)	O. U. Early primary atrophy of optic nerve, more marked in O. D., and possibly localized somewhat in papillomacular bundles	O. D. 20/30—3 O. S. 20/20+1			
53	O. D. Defect in upper temporal field O. S. Blind (old injury)	O. D. Moderate primary atrophy of optic nerve O. S. Advanced primary atrophy of optic nerve	O. D. 20/40 O. S. Blind	Tabes	Followed for 4 yr.; no significant change	Congenital red-green color blindness
54	.....	.....	.....	Diffuse meningovascular	Followed for 6 yr. 8 mo.; defects in field became gradually worse between February and September 1932; no further decrease between September 1932 and April 1937; condition worse in October 1937	Atrophic choroiditis in O. S.
2/15/32	O. D. Peripheral contraction, especially of nasal field O. S. Not tested (atrophic choroiditis)	O. D. Early primary atrophy of optic nerve	O. D. 20/20			
5/20/32	O. D. Peripheral contraction, especially of nasal field and cecocentral scotoma for 1° blue	O. D. Moderate primary atrophy of optic nerve	O. D. 20/50			
9/21/32	O. D. Peripheral contraction as previously described and cecocentral scotoma for 1° white (fig. 16)	O. D. Advanced primary atrophy of optic nerve	O. D. 20/200			
55	.....	.....	.....	Tabes	Followed for 4 yr.; central scotoma developed during first year; no further change in scotoma; peripheral field showed gradual improvement	O. D. Blind (atrophic choroiditis, retinitis proliferans)
5/31/33	O. D. Blind (atrophic choroiditis, retinitis proliferans) O. S. Concentric peripheral contraction for 1° white; 1° blue and red not seen	O. S. Early primary atrophy of optic nerve	O. S. 20/40—1			
4/13/34	O. S. Relative central scotoma for ½° white; peripheral limits show improvement (fig. 17)	O. S. Advanced primary atrophy of optic nerve	O. S. 20/200+1			
56	.....	.....	.....	Dementia paralytica	Followed for 2 yr.; fields became worse	Congenital red-green color blindness
2/13/35	O. U. Peripheral contraction, particularly in upper and lower meridians	O. U. Advanced primary atrophy of optic nerve	O. D. 20/100 O. S. 20/70			
3/19/37	O. U. Increased contraction and involvement of foveal regions (fig. 18)	O. U. Advanced atrophy, especially of papillomacular bundle	O. D. 20/100 O. S. 20/70—2			

showed bilateral atrophy confined to the papillomacular bundle. In case 37 there was a cecocentral scotoma of the left eye combined with a peripheral defect most marked in the upper field. The 1 degree red test object was seen in a small area in the lower field. Because of the advanced field changes in the right eye and the unsteady fixation, the results are somewhat unreliable. The findings indicate, however, that this eye also had a central scotoma combined with a defect in the periph-

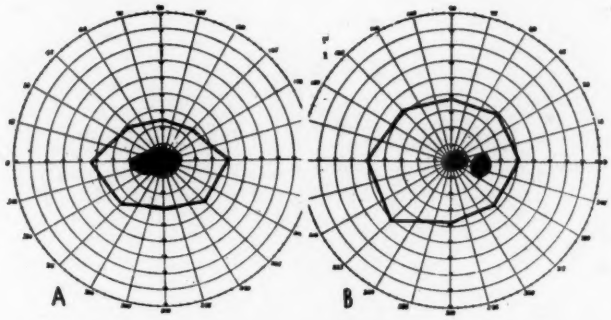

Fig. 7 (case 35).—Visual field (*A*) of left and (*B*) of right eye in a case of advanced atrophy, especially of the papillomacular bundle, showing cecocentral scotomas and concentric contraction (type 4). Solid areas indicate absolute and cross-hatched areas relative scotoma for a ½ degree white test object. Vision of the left eye was 2/200 and of the right 10/200.

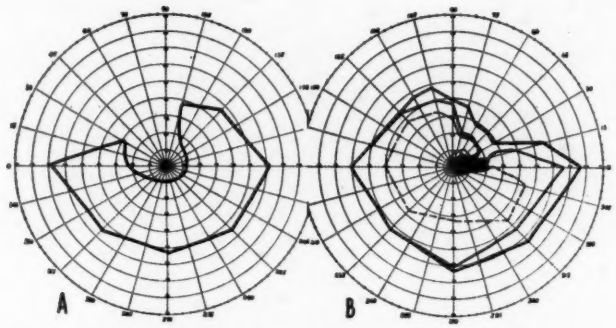

Fig. 8 (case 36).—Visual field (*A*) of left and (*B*) of right eye in a case of advanced atrophy of the papillomacular bundle. For the left eye, vision was 2/200, and the field outlined is that for the 1 degree white test object. For the right eye, vision was 20/200, and the solid area indicates scotoma for a 1 degree white test object; the broken line bounds the field for the 1 degree red, the inner solid line that for the ½ degree white and the outer solid line that for the 1 degree white test object.

eral field. The visual acuity was 18/200 in the right eye and was limited to perception of hand motions in the left eye. The disks showed primary atrophy, more marked in the papillomacular bundle.

In cases 38, 39 and 40 the fields for the right eyes only are shown, as the vision of the left eye in each case was limited to light perception. In case 38 the central area showed involvement for colors but not for the 1 degree white test object. A cecocentral scotoma was mapped with a 1 degree blue test object. The 1 degree red object was seen only in a small area in the nasal field. The 1 degree white test object showed

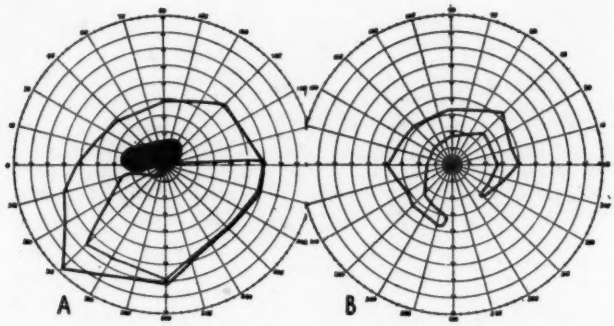

Fig. 9 (case 37).—Visual field (*A*) of left and (*B*) of right eye in a case of advanced atrophy, especially of the papillomacular bundle. In *A*, the solid area is a scotoma for a 1 degree white test object; the outer line bounds the field for a 3 degree white and the inner line that for a 1 degree white test object. The field in *B* is for a 3 degree white test object. Vision of the left eye was 18/200, and that of the right was limited to the perception of hand movements.

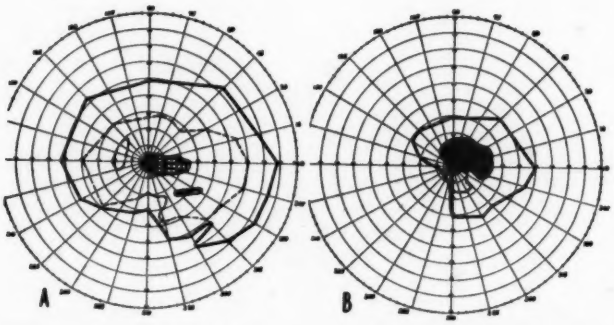

Fig. 10.—Right visual field in 2 cases of advanced primary atrophy of the optic nerve (*A*, case 38; *B*, case 39), showing cecocentral scotomas and peripheral defects (type 4). In *A*, the field bounded by the solid line is that for a 1 degree white test object; that bounded by long dashes, for a 1 degree blue object, and that bounded by short dashes, for a 1 degree red object. Cross-hatched areas indicate scotomas for the 1 degree blue test object. In *B*, the outer line bounds the field for a 3 degree white test object, and the inner line, that for a 1 degree white test object. The solid area indicates a scotoma for a 3 degree white test object. Vision in *A* was 20/200 and in *B* 7/200.

a defect limited to the lower field. The visual acuity was 20/200 in the right eye and was reduced to light perception in the left. The disks showed generalized pallor, more advanced in the left eye. In case 39

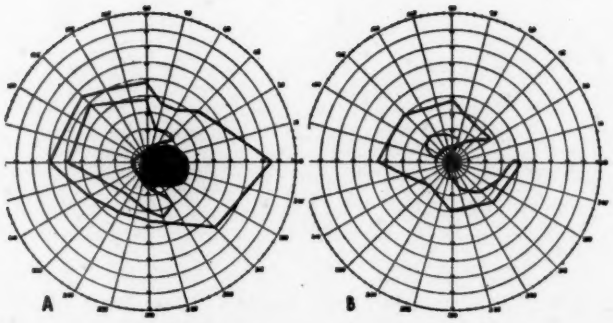

Fig. 11 (case 40).—Visual field of right eye in a case of advanced primary atrophy of the optic nerve (*A*) on Nov. 2, 1932, and (*B*) on Sept. 12, 1934, showing progressive changes in a defect in field of type 4. The outer line bounds the field for a 3 degree white and the inner line that for a 1 degree white test object. The solid area indicates a scotoma for a 3 degree white test object. Visual acuity at the time of the first determination was 20/200 and at the second 8/200.

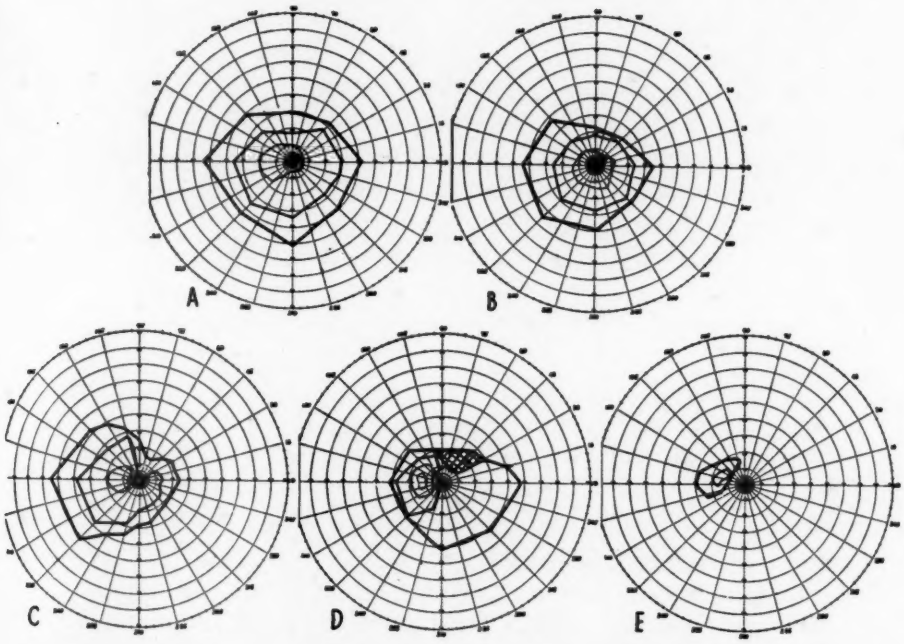

Fig. 12 (case 41).—Progressive changes in a type 4 defect in the right visual field in a case of advanced atrophy, especially of the papillomacular bundle. On Dec. 20, 1935, and on June 19, 1936 (*A* and *B*), vision was 20/200; on Jan. 12, 1937 (*C*), 10/200, and on April 28 and on Nov. 2, 1937 (*D* and *E*), 4/200. In *A*, *B* and *C*, the outer solid line bounds the field for a 3 degree white, the inner solid line that for a 1 degree white and the broken line that for a 1 degree red test object. The cross-hatched area in *A* and in *B* represents scotoma for a 1 degree red test object. In *D*, the broken line bounds the field for a 1 degree red test object and the solid lines, progressing from the center toward the circumference of the illustration, bound the fields for ½ degree white, 1 degree white and 3 degree white test objects. Relative scotoma for a 3 degree white test object is represented by the cross-hatched area. In *E* the outer line bounds the field for a 3 degree white and the inner line that for a 1 degree white test object. A ½ degree white and a 1 degree red test object were not seen.

the right eye showed an advanced defect. Perception of color was entirely lost. The 1 degree white object was seen only in a small area in the lower temporal field. Tests with a 3 degree white test object showed a large cecocentral scotoma and marked peripheral contraction. The visual acuity was 7/200 on the right and was reduced to perception of hand motions on the left. In case 40 there was an advanced defect in the field of the right eye, with complete loss of color vision. The 3 degree white test object showed a large central scotoma and moderate peripheral contraction. A retest of the fields about two years later showed a slight increase in the extent of the scotoma and a marked decrease in the peripheral limits. The results on both dates indicated a greater involvement of the temporal than of the nasal field. The visual acuity of the right eye was 20/200 at the time of the first test and 8/200 at the time of the second. Although perimetric studies of the left eye were impossible, it is interesting that on the first date a 3 degree white test object was seen doubtfully in the nasal field only, and on the second date perception of light was confined to the nasal field. These findings indicate that the course of development in the left eye was probably similar to that in the right. The patient showed advanced generalized primary atrophy of the optic nerve in both eyes.

The visual fields in case 41, shown for the right eye only, illustrate the progressive changes in a somewhat similar defect. The disks showed marked primary atrophy of the optic nerve, especially in the area of the papillomacular bundle. In December 1935 and in June 1936 there was a central scotoma for a 1 degree red but not for a 1 degree white test object. In January 1937 a 1 degree white object was not seen at the fovea and the field for the 1 degree red object was reduced to a small area in the nasal field. In April 1937 the extent of the field for a ½ degree white object was approximately equal to the earlier field for a 1 degree red object. The charts illustrate the way in which defects in the field for color and for small white test objects frequently predict the defects which may later be found for larger white test objects if the progress of the atrophy is not arrested.

In 5 cases defects of type 4 in the earlier stages were similar to the nerve fiber bundle defects seen in cases of glaucoma and in some cases of choroiditis juxtapapillaris. The progressive changes in the fields in 3 of the 5 cases (48, 49 and 50) are shown in figures 13 to 15. In case 48, at the time of the first examination, a 1 degree white test object showed a scotoma extending nasally and a sector-shaped defect in the lower field giving a large nasal step. The areas blind to the 1 degree white object did not include the fixation point in either eye. Tests with a 1 degree blue object (made only of the right eye) showed a defect extending from the blindspot to the nasal periphery and involving the fixation point. A 1 degree red object was

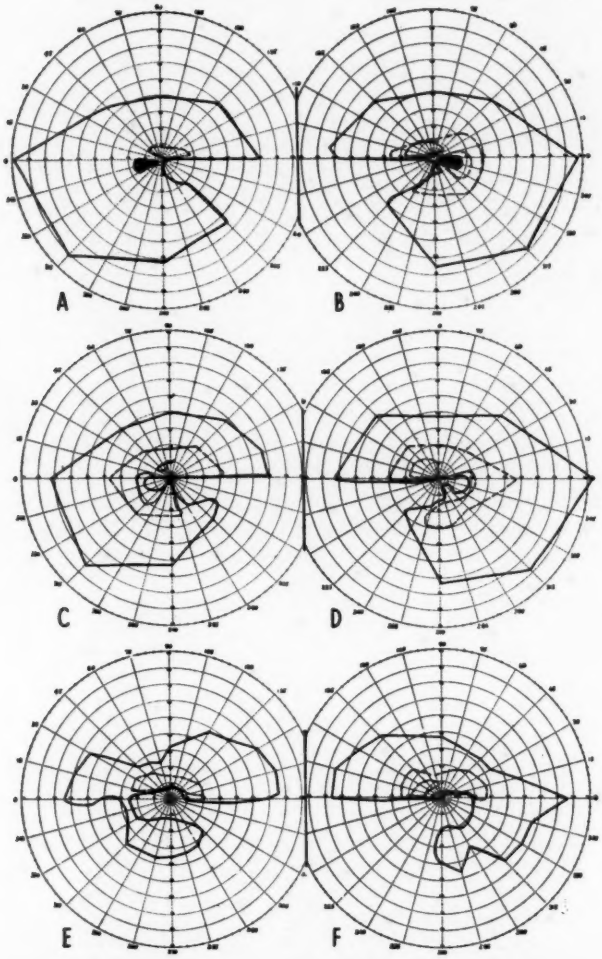

Fig. 13 (case 48).—Progressive changes in nerve fiber bundle defects (type 4). On June 28, 1933, moderate primary atrophy of the optic nerves was present and vision in the left (*A*) and the right (*B*) eye was 20/50 —1. The solid area in the map of each field at that time represents scotoma for a 1 degree white test object, and the solid line, the boundary of the field for the same object. The broken line in *A* bounds the field for a 1 degree red object, as does the inner broken line in *B*. The outer broken line in *B* bounds the field for a 1 degree blue test object. On Oct. 31, 1933, and on Feb. 24, 1936, the atrophy was in an advanced stage, the vision of each eye being 20/200 on the earlier date and 4/200 on the later one. In *C* and *D*, representing the left and the right eye, respectively, on the earlier date, the solid line bounds the field for a 1 degree white test object; the outer broken line, that for 1 degree blue, and the inner broken line, that for 1 degree red. In *E* and *F*, representing the left and the right eye on the later date, the solid line bounds the field for the 1 degree white and the outer broken line that for the 1 degree blue test object. The inner broken line in *F* bounds the field for a 1 degree red test object (not visible to the left eye at that time).

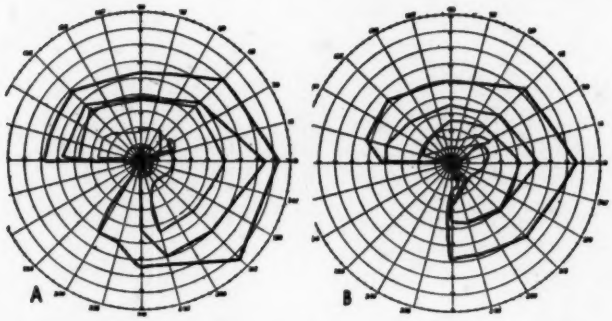

Fig. 14 (case 49).—Right visual field (*A*) on Feb. 21, 1935, and (*B*) on Sept. 9, 1936, showing progressive changes in nerve fiber bundle defects (type 4). Moderate atrophy of the optic nerve, especially of the papillomacular bundle, was present at the time of the first observation, and vision was 10/200; at the time of the second observation, primary atrophy of the optic nerve was advanced, and vision was 16/200. A 3 degree red test object was not seen at either time. Progressing centrally from the circumference of the illustration, the solid lines bound the fields for the 3 degree, the 1 degree and the ½ degree white test object; the broken line bounds the field for the 1 degree blue test object.

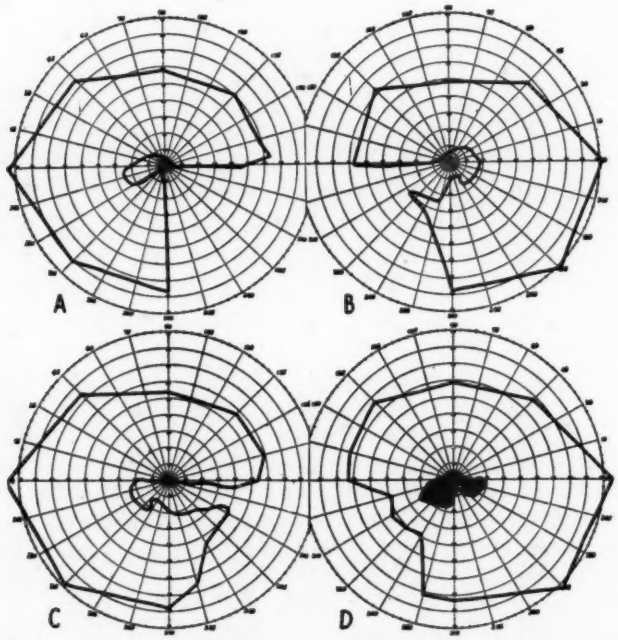

Fig. 15 (case 50).—Defects of nerve fiber bundle type (type 4), showing improvement in one month in a case of moderate primary atrophy of the optic nerve. *A* and *B* show the left and the right field, respectively, on March 8, 1937, for a 1 degree white test object. Vision was 5/200 for the left and 3/200 for the right eye. *C* and *D* show the left and the right field on April 2 for a 1 degree white test object. The solid area in the right field represents scotoma for a 1 degree white test object. Vision was 7/200 for the left and 5/200 for the right eye.

seen only in a small area in the upper nasal field of each eye. At this time the visual acuity was 20/15—1 in both eyes, and ophthalmoscopic examination showed moderate primary atrophy of the optic nerve. Four months later the regions blind to the 1 degree white object had enlarged and included the fixation point. The visual acuity had decreased to 20/200, and both nerve heads showed advanced atrophy. The last perimetric examination, about two years later, showed, in addition to an increase in the central defect, considerable peripheral loss in all but the upper nasal sector of the field. A 1 degree red object was seen only in a small area in the upper nasal field of the right eye and not at all in the left eye. The visual acuity was 4/200 in both eyes. In these fields, as in those of case 41, the early findings with 1 degree blue and red test objects indicated the future course of the changes for the 1 degree white test object.

In case 49 a defect in the right eye extended from the blindspot into the lower nasal field, producing a nasal step. At the time of the first examination the tests with a 1 degree white object showed no evidence of involvement of the upper and temporal regions. The 1 degree blue test object was seen only in the upper field, however, and the perception of red was lost entirely. In the left eye the 1 degree white object was seen only in the upper nasal field. (Fields for this eye are not shown in the charts.) The vision was 10/200 in the right eye and 6/200 in the left. The disks showed well developed atrophy, especially marked in the area of the papillomacular bundle. Tests made about eighteen months later showed a loss in the upper and temporal portions of the peripheral field of the right eye but little increase in the defect in the lower nasal field. The visual acuity was essentially unchanged. In case 50 the fields showed a similar nerve fiber bundle defect involving the cecocentral area and the lower nasal field. The visual acuity at the time of the first test was 3/200 in the right eye and 5/200 in the left. The disks showed bilateral primary atrophy of the optic nerve, more marked in the right eye. Reexamination of the fields one month later showed some improvement in the defects in the lower nasal quadrants. The visual acuity of the right and of the left eye were 5/200 and 7/200, respectively.

The changes in the fields of the 3 patients in whom central scotoma developed after the peripheral defects are shown in figures 16 to 18. In case 54 the field of the right eye only is shown; atrophic choroiditis was present in the left eye. In the right eye at the time of the first examination there was a contraction of the nasal field for 1 degree white and colored objects. The blindspot for a 1 degree red test object showed a slight enlargement toward the fixation point. The visual acuity at this time was 20/20—. The right nerve head showed early generalized primary atrophy. Three months later a cecocentral scotoma for a 1 degree blue object merged with the defect in the nasal

field. The vision had decreased to 20/50, and the disk showed a moderate degree of pallor. Four months later there were beginning cecocentral scotoma and slightly greater nasal contraction for a 1 degree white object and complete loss of field for a 1 degree red object. The visual acuity had fallen to 20/200, and ophthalmoscopic examination

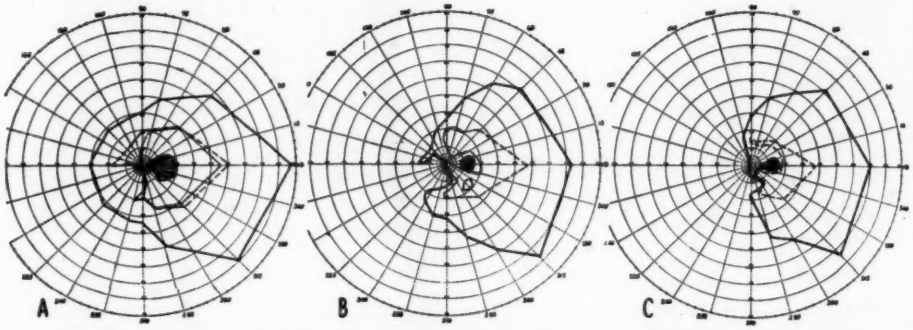

Fig. 16 (case 54).—Visual field of the right eye, showing development of cecocentral scotoma in association with (*A*) early, (*B*) moderate, and (*C*) advanced primary atrophy of the optic nerve. Observations were made on Feb. 15, May 20 and Sept. 21, 1932, when vision was 20/20, 20/50 and 20/200, respectively. The cross-hatched area represents scotoma for a 1 degree red test object, and the solid areas, scotoma for a 1 degree white test object. The solid line bounds the field for the 1 degree white test object; the line of long dashes, that for the 1 degree blue, and the line of short dashes, that for the 1 degree red (not seen at the time of the last observation).

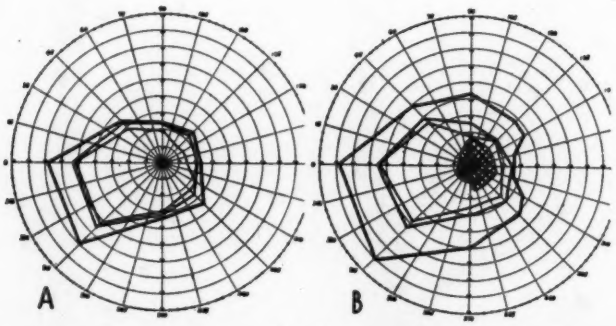

Fig. 17 (case 55).—Visual field of the left eye, showing development of central scotoma after peripheral defect in association with (*A*) early and (*B*) advanced primary atrophy of the optic nerve. Progressing centrally from the circumference of the illustration, the solid lines bound the fields for the 3, the 1 and the ½ degree white test object. Observations were made on May 31, 1933, and April 13, 1934, when the vision was 20/40 —1 and 20/200 +1, respectively. The cross-hatched area indicates relative scotoma for the ½ degree white test object.

showed advanced primary atrophy. The defects for 1 degree red and blue objects predicted the future loss in the field for the 1 degree white object.

In case 55 the field of the left eye only is shown; atrophic choroiditis was present in the right eye. At the time of the first examination the left eye showed a definite decrease in the extent of the fields for 3, 1 and ½ degree white test objects. One degree blue and red objects were not seen in any part of the field. The corrected visual acuity was 20/40—1. The disk showed incipient generalized primary atrophy. Retests approximately ten months later showed some improvement in the peripheral limits of the field for 3 degree white and essentially unchanged fields for 1 and ½ degree white objects. A large relative scotoma for a ½ degree white test object had developed in the central

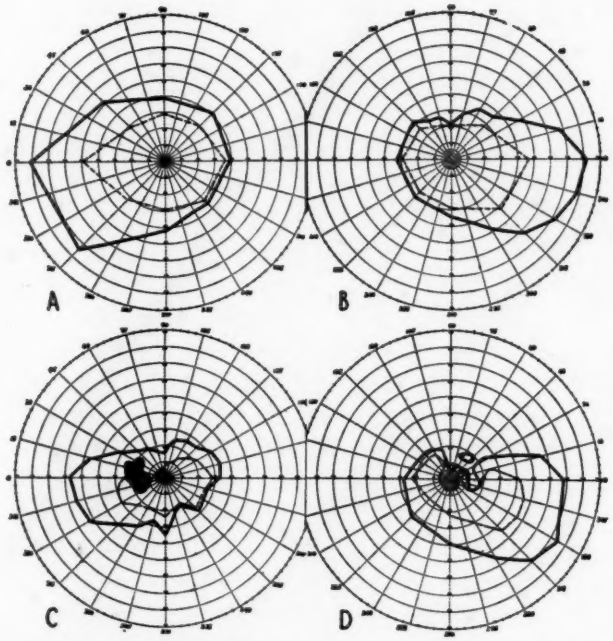

Fig. 18 (case 56).—Progression of peripheral contraction and development of central scotomas in a case of advanced primary atrophy of the optic nerve. The fields for a 1 degree white test object are bounded by a solid line and those for a 1 degree blue test object by a broken line, *A* and *B* showing the left and the right field of Feb. 13, 1935, and *C* and *D* those of March 19, 1937. In *C* the solid area represents scotoma for a 1 degree white and the cross-hatched area scotoma for a ½ degree blue test object. Vision at both determinations was 20/70 for the left and 20/100 for the right eye. Atrophy was pronounced in the papillomacular bundles at the time of the second determination.

field. The visual acuity had dropped to 20/200 + 1, and the disk showed advanced primary atrophy.

In case 56 at the time of the first examination contracted fields for the 1 degree white object were found in both eyes. The fields for the

1 degree blue object were of normal extent. The 1 degree red object appeared white, but there was a possible congenital red-green color blindness, so that the findings with this test object were of doubtful value. The visual acuity was 20/100 in the right and 20/70 in the left eye. This marked reduction in acuity indicated involvement of the central field and suggested a central defect for red due to the atrophy of the optic nerve, in addition to the probable congenital defect, since the latter does not cause reduction in acuity. The nerve heads at this time showed advanced generalized primary atrophy. Retests of the fields two years later showed greater contraction, particularly in the upper temporal field. In the right eye the defect for blue extended downward to include the foveal region. In the left eye the field for a 1 degree blue test object showed a contraction of the limits of the upper temporal region to within about 6 degrees of the fixation point. There was, in addition, a central scotoma for a ½ degree blue test object. The visual acuity was 20/100 in each eye. The disks on this date showed especially marked pallor in the area of the papillomacular bundles.

Comment.—Figures 1 to 18 represent the four general types of defect found in the entire group of 56 patients with syphilitic primary atrophy of the optic nerve. Although the field defects are of different forms, there are certain characteristics common to the entire group. The fields of the two eyes show, as a rule, a marked similarity, although the defects are frequently more advanced in one eye than in the other. For example, some patients have bilateral central scotomas; others have bilateral concentrically contracted fields or localized peripheral defects in both eyes. Moreover, when bilateral localized peripheral defects are present, corresponding regions of the two fields are affected. In case 48 (fig. 13), for example, a scotoma extended from the blindspot to the nasal periphery in the lower field of each eye. In other cases the similarity of the changes in the two eyes results in heteronymous hemianopic defects, which may be binasal, bitemporal or altitudinal. With only 1 exception (case 44), all patients with atrophy involving both optic nerves who retained sufficient vision in each eye to permit accurate perimetric study showed this similarity of defects.

The nature of the progressive decrease in the visual fields is illustrated in cases 1, 7, 29, 40, 41, 48, 49, 54, 55 and 56 (figs. 1, 3, 6, 11 to 14 and 16 to 18). Defects for red practically always precede those for blue, as is the rule with lesions of the conducting pathways. The results obtained on any one date with a graded series of test objects often give information not only of the extent of involvement but of the future course of the field defect. In case 54, for example, the cecocentral scotoma for the 1 degree red object found at the first examination preceded a similar scotoma for the 1 degree blue and

later for the 1 degree white object. Similarly, in case 41 there was a central scotoma for the 1 degree red object only at the time of the first examination. One year later this central defect was demonstrable with the 1 degree white test object. In case 48, likewise, the fields for the 1 degree white object showed at first only a localized defect, but the results obtained with the 1 degree red object showed widespread involvement.

Forty of the 56 patients were reexamined at later dates. Twelve, or 30 per cent, pursued a steady downhill course, 3 becoming totally blind and 9 showing a steady loss in the visual fields. Seventeen showed unchanged fields during the period of observation. Six showed fluctuations in the fields, while 5 (cases 3, 8, 16, 36 and 50) showed definite improvement, 1 regaining normal fields. Thus, during the period of observation 28, or 70 per cent, showed at least a temporary arrest in the progress of their optic neuropathy. The efficacy of various forms of antisyphilitic therapy in arresting the progress of atrophy of the optic nerve will be the subject of later reports.

The perimetric fields in cases 8 and 16 (figs. 4 and 5) illustrate the extent of improvement which may occur. In case 16 the change consisted only in a slight widening of the field for blue, but since this improvement involved the fixation point, it resulted in an increase in visual acuity from 20/70 + to 20/40 — 1. In case 8 a slight defect in the peripheral field for 1 degree white and colored objects disappeared entirely.

From the data for the entire group, summarized in tables 1 to 4, conclusions may be drawn as to the relative frequency of the different forms of field defect and the relation between the type of defect and the type of neurosyphilis, the type of atrophy of the optic nerve and the loss in visual acuity.

Central scotoma (type 3 or 4 defect) occurred in 30 of the 56 patients (53 per cent). No apparent relation exists between type of neurosyphilis and type of field defect. The 56 patients included 30 who had parenchymatous neurosyphilis (tabes, dementia paralytica or the tabetic form of dementia paralytica), 22 who had meningovascular neurosyphilis, 2 who had congenital neurosyphilis and 2 who had latent syphilis of the central nervous system and seronegative cerebrospinal fluid. Forty-seven per cent of the patients who had parenchymatous neurosyphilis and 64 per cent of those who had meningovascular syphilis had central scotoma, i. e., a field defect of type 3 or 4.

There is a definite relation between the type of atrophy of the optic nerve and the type of field defect. Especial involvement of the papillomacular bundle was noted in many of the patients with a defect of type 3 or 4 but was not observed in patients with a defect of type 1 or 2. Only 2 of the 8 patients with central scotomas and normal

peripheral fields (type 3 defect) showed generalized atrophy of the nerves. The remaining 6 patients who had this type of defect showed atrophy limited almost entirely to the papillomacular bundles. Seven of the 22 patients with central scotomas combined with peripheral defects (type 4 involvement) showed a greater degree of atrophy in the areas of the papillomacular bundles than in the remaining portions of the nerves. Thus, central scotoma is invariably present in patients who show distinctly localized atrophy of the papillomacular bundle, and from this clinical finding the presence of such scotoma may be prophesied. In patients with generalized pallor of the nerve, however, clinical examination gives no information as to the presence or absence of central scotoma.

A number of the patients with field defects of type 1 or 2 had normal visual acuity, of 20/15, in one or both eyes. For those with defects of type 1 the vision was 20/15 in 7 of the 14 eyes. For those with type 2 defects the vision was 20/15 in 5 of the 36 affected eyes. Only 1 instance of normal visual acuity was observed among the 30 cases of central or cecocentral scotoma. In this case (39) the scotoma in the right eye extended close to but did not involve the fixation point.

A study of the relationship between visual acuity, ophthalmoscopic findings, and visual fields in the 56 cases suggests that the changes in the fields are probably the earliest clinical evidence of atrophy of the optic nerve. Defects in the visual fields were found for all patients who showed any reduction in the corrected visual acuity. However, a number of patients, had normal acuity in spite of both defects of type 1 or 2 in the fields and pallor of the nerve heads. Field defects were found in every patient who showed pallor of the disks on ophthalmoscopic examination, a number of those showing only early pallor having fairly well advanced defects in the fields. Three patients who had normal nerve heads at the time of the first ophthalmoscopic examination and subsequently had definite atrophy of the optic nerves showed defects in fields at the time of the first examination. It seems evident, therefore, that changes in the visual fields may antedate the ophthalmoscopic appearance of atrophy and that both may precede loss of central vision.

After a diagnosis of primary atrophy of the optic nerve has been made, studies of the visual fields provide a more reliable means of following the course of the atrophy than does either an ophthalmoscopic examination or a determination of the visual acuity. Since it is difficult to differentiate more than three stages of pallor of the disk, it is obvious that moderate progressive changes in the atrophic process may not be detected by ophthalmoscopic examination. The visual acuity may likewise remain unchanged while definite alterations in the visual field are taking place. The impairment of visual acuity depends, as a rule, entirely on the involvement of the fibers of the papillomacular bundle,

which supplies the fovea. When, however, the foveal impairment is sufficient to reduce the central acuity to 20/100 or less, the patient may use slightly eccentric fixation, so that further decrease in visual acuity is dependent on a change in the field in the paracentral region. In either case the maintenance of visual acuity at a constant level for a considerable period by no means indicates that the progress of atrophy of the optic nerve is arrested. In case 7, for example, between January 1932 and February 1936 successive perimetric tests (fig. 3) showed an increasing contraction, but no change was evident in the ophthalmoscopic appearance or in the visual acuity. In case 29 the increase in the scotoma in the right eye (fig. 6) was accompanied with an increase in pallor, but because the fovea was not involved normal visual acuity was maintained.

(b) *Patients with Defects in the Fields Associated with Normal Optic Nerve Heads.*—Perimetric examination of 200 neurosyphilitic patients whose optic disks were normal or showed only a questionable pallor disclosed in 12 cases definite slight defects in the fields which could not be attributed to poor cooperation on the part of the patient or to other complicating factors. The clinical findings in these 12 cases are summarized in table 5.

Six patients had bilateral field defects, and 3 had unilateral defects. In the remaining 3 only one eye was tested, because of complicating factors in the second eye. Three patients showed a questionable pallor of one or both nerve heads, while the eyes of 9 appeared entirely normal on ophthalmoscopic examination.

Four patients (cases 57 to 60) had slight peripheral field defects of type 1. The left field of 1 of these patients (case 57) is shown in figure 19. Five patients (cases 61 to 65) had slight defects of type 2. Figure 20 shows the fields of 2 of these patients. In 1 a nerve fiber bundle defect produced an alteration resembling that of type 4 but differing in that there was no involvement of the cecocentral region. Seven of the 9 patients with slight peripheral defects of type 1 or 2 had normal visual acuity with correction; no refraction was made for the other 2, and the slightly reduced vision, of 20/30, was presumably due to an error of refraction.

Two patients (cases 66 and 67) had slight defects of type 3. One of these had an absolute central scotoma for ½ degree red and blue test objects and vision of 20/30. The other had only a relative scotoma for ½ degree colored objects and 20/20 vision. One patient (case 68) had a field defect of type 4 in the left eye. The right eye was not tested because of incipient cataract. The field of the left eye on three determinations is shown in figure 21.

Comment.—Seven of these 12 patients were followed for from three months to five years. Five were examined only once and then

TABLE 5.—*Patients with Defects in Fields and Normal Nerve Heads*

Case No. and Date Examined	Observations in Visual Fields	Appearance of Nerve Heads	Corrected Visual Acuity	Type of Syphilis	Progressive Changes in Visual Fields	Comment
57	.....	.....	.....	Diffuse meningo-vascular	Followed for 5 yr. 9 mo.; gradual decrease in fields	O. D. Ophthalmoplegia interna; perimetric findings variable because of blurring of near vision
3/14/31	O. D. Slight concentric contraction (results variable) O. S. Slight but reproducible concentric contraction	O. U. Normal				
11/10/31	O. D. Not tested O. S. Further contraction	O. U. Normal				
2/ 1/36	O. S. Further slight decrease					
12/18/36	O. S. Further slight decrease (fig. 19)	O. U. Normal	O. D. 20/20+3 O. S. 20/15—3			
58	O. U. Slight concentric contraction for 1° white	O. D. Questionable pallor O. S. Normal	O. D. 20/15 O. S. 20/15—1	Dementia paralytica	Followed for 2 yr. 8 mo.; no change	
59	O. U. Slight concentric contraction	O. U. Normal	O. D. 20/15—1 O. S. 20/15	Tabes	Followed for 9 mo.; no change	
60	O. U. Slight concentric contraction	O. U. Questionable pallor of disks	O. U. 20/15	Diffuse meningo-vascular	Followed for 2 yr. 6 mo.; fields improved, became worse and improved again in successive tests	
61	O. D. Not tested O. S. Slight cut in lower nasal field (fig. 20)	O. U. Normal	O. D. 20/50—2 (amblyopic eye) O. S. 20/20	Asymptomatic of central nervous system; late	Followed for only 3 mo.; no change	O. D. Amblyopia; congenital strabismus
62	O. D. Nerve fiber bundle defect; macular region spared (fig. 20) O. S. Normal	O. U. Questionable pallor of disks	O. D. 20/20— O. S. 20/30 (uncorrected refractive error)	Tabes	Seen only once	
63	O. D. Normal O. S. Slight temporal contraction	O. U. Normal	O. D. 20/15 O. S. 20/15	Asymptomatic of central nervous system; late	Seen only once	
64	O. U. Slight nasal contraction	O. U. Normal	O. D. 20/15 O. S. 20/15	Asymptomatic of central nervous system; late	Seen only once	
65	O. D. Normal O. S. Slight temporal contraction	O. U. Normal	O. D. 20/20 O. S. 20/30 (uncorrected)	Diffuse meningo-vascular	Seen only once	
66	O. U. Relative central scotomas for ½° blue and red; normal peripheral fields	O. D. Normal O. S. Normal	O. D. 20/20—3 O. S. 20/20+3	Tabes	Followed for 4 yr.; no change	
67	O. U. Absolute central scotomas for ½° blue and red; normal peripheral fields	O. D. Normal O. S. Normal	O. D. 20/30 O. S. 20/30—1	Diffuse meningo-vascular	Seen only once	
68	.....	.....	.....	Diffuse meningo-vascular	Followed for 5 yr. 8 mo.; complete recovery	O. D. Opacity of lens
5/5/31	O. D. Not tested (lens opaque) O. S. Relative cecocentral scotoma for ½° white; concentric peripheral contraction	O. S. Normal	O. S. 20/40 (uncorrected)			
11/15/32	O. S. Improvement	O. S. Normal				
2/15/35 to 1/31/36	O. S. Normal (fig. 21)	O. S. Normal	O. S. 20/15 corrected; 20/40 uncorrected			

were lost from observation. Since none of the patients followed has as yet shown unquestioned pallor of the disks, definite proof is lacking that the field defects were due to incipient atrophy. Four other patients in whom primary atrophy later developed but in whom the

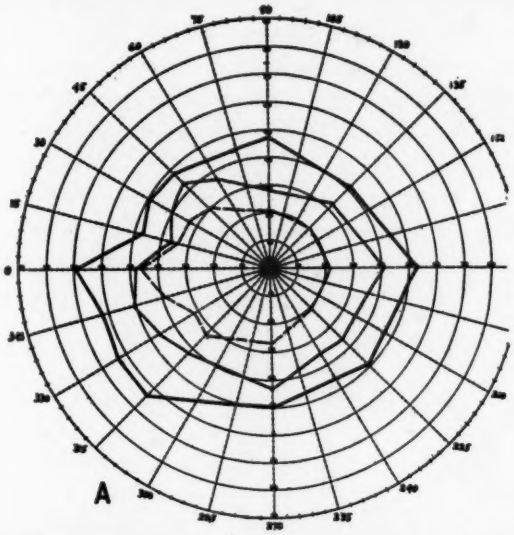

Fig. 19 (case 57).—Visual field of the left eye on Dec. 18, 1936, showing a defect of type 1. The broken line bounds the field for the 1 degree red test object; the inner solid line, that for the ½ degree white object, and the outer solid line, that for the 1 degree white object.

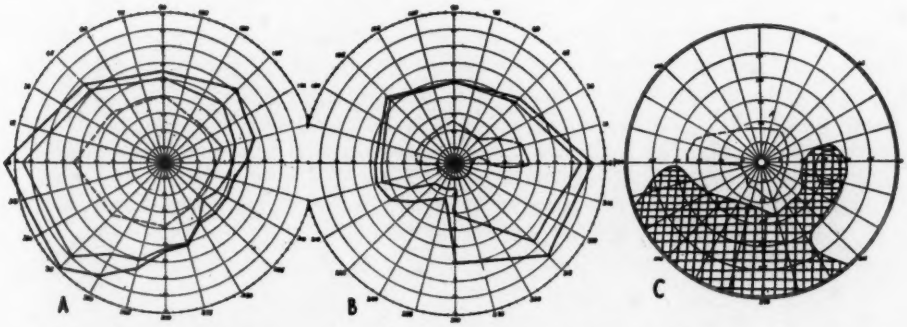

Fig. 20.—Defects of type 2. The broken line in *A* (case 61) bounds the visual field of the left eye for a 1 degree red test object; the inner solid line, that for a ½ degree white object, and the outer solid line, that for a 1 degree white object. Visual acuity was 20/20. The lines in *B* (case 62) bound the right field for the same test objects. Visual acuity was 20/30— (uncorrected). The broken line in *C* bounds the field for a ½ degree red test object in the same case, and the cross-hatched area represents scotoma for a ½ degree gray test object.

field defects in one or both eyes antedated pallor of the disks have already been reported on under the section dealing with primary atrophy (cases 1, 2, 3 and 52). Since the field defects reported here,

though slight, conform to the types found with established primary atrophy of the optic nerve, it seems justifiable to attribute these early defects to incipient primary atrophy of the optic nerve which is not yet manifest on ophthalmoscopic examination.

The outcome in the 7 cases in which prolonged observation was possible was as follows: One patient with a type 1 defect showed a gradual loss in the fields during five years' observation. The vision and the fundi remained normal. Four patients showed no change in fields, fundi or vision during periods ranging from three months to four years. The patient with a type 4 defect in the left eye showed improvement in the field and in vision when retested after six months and complete recovery when retested four and five years after the first examination (fig. 21). The nerve head remained normal throughout the period of observation. One patient who had field defects of type

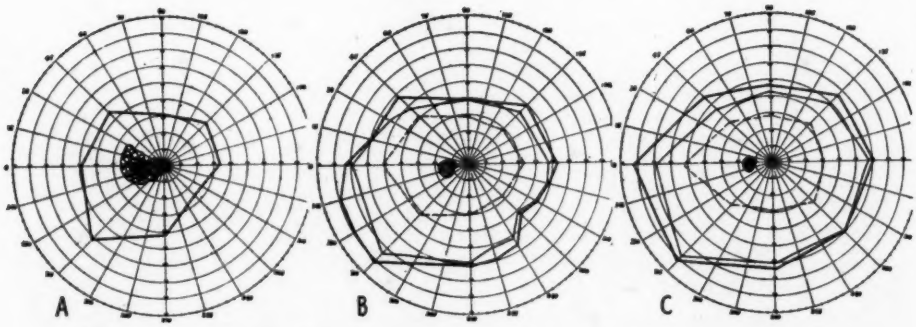

Fig. 21 (case 68).—Left visual field on (*A*) May 5, 1931, (*B*) Nov. 15, 1932, and (*C*) Feb. 15, 1935, showing improvement in a field defect of type 4. At the first examination vision was 20/40. The solid area represents a blindspot for a ½ degree white test object, and the cross-hatched area, relative scotoma for the same object. The field illustrated is that for a 1 degree white test object. At the second examination vision was 20/15. The cross-hatched area represents a blindspot for a ½ degree red test object; the broken line bounds the field for a 1 degree red test object, the inner solid line that for a ½ degree white test object and the outer solid line that for a 1 degree white test object. At the time of the third examination vision was 20/15. The cross-hatched area represents a blindspot for a ½ degree red test object. The lines bound fields for the same test objects in *C* as in *B*.

1, normal vision and questionable pallor of the disks showed fluctuating changes in the fields, with both increase and decrease in the limits on different examinations. There was no change in the appearance of the nerve heads. The possible effect of antisyphilitic therapy on the progress of the field defects will be considered in a later paper.

Central scotoma was observed in 3 of these 12 patients with defects in the fields and no manifest ophthalmoscopic evidence of atrophy (cases 66, 67 and 68). This is an incidence of only 25 per cent, against one

of over 54 per cent for patients with manifest atrophy. This may indicate that involvement of the papillomacular bundle tends to occur late rather than early in the development of the atrophy.

(c) *Patients with Homonymous Defects in the Fields.*—Homonymous defects in the fields were found in 12 of 291 patients with syphilis of the central nervous system. These homonymous defects were partial or complete hemianopia, homonymous quadrantic defects, homonymous scotomas and, in 1 case, double quadrantic defect. Figures 22, 23 and 24 illustrate these changes, and the clinical findings are epitomized in table 6.

Comment.—The pertinent questions in these cases of homonymous defects in the fields are: 1. Are such defects to be attributed to lesions involving both optic nerves anterior to the chiasm or to lesions in the tracts or the optic radiations posterior to the chiasm? 2. Are the actual lesions due to syphilitic infection or to some other cause? A study of these 12 cases indicates that the lesions responsible for the visual defects lay posterior to the chiasm and were not definitely related to the syphilitic infection. One patient had a definite medical record of hemiplegia; 2 patients had a record of severe cranial traumatism, and 7 patients had a history indicative of hemiplegia, aphasia or other disease of the central nervous system. These 10 patients showed, variously, normal or questionable pallor of the optic nerves and definite sclerosis of the retinal vessels. In no case could a diagnosis of even early atrophy of the optic nerve be made. The evidence in these 10 cases indicates that the defects in the fields are probably due to a cerebral injury posterior to the chiasm, secondary to either trauma or intracranial hemorrhage.

The 2 remaining patients with homonymous defects in the fields (cases 82 and 83) had no history and showed no evidence of a vascular lesion, a cerebral injury or other complicating factor. Both had Argyll Robertson pupils. One had normal fundi; the other showed advanced primary atrophy of the optic nerve of the right disk and questionable pallor of the left. There was no evidence to indicate the cause of the hemianopia of these 2 patients. However, in view of the fact that there was positive evidence of cerebral damage in 10 patients with similar involvement, it appears probable that intracranial hemorrhage or a localized area of softening affecting the visual pathways was likewise responsible for the hemianopia of these 2 patients. The visible atrophy of the optic nerve of the 1 patient could well have been due to an independent syphilitic process in the optic nerve. That this is the probable explanation is indicated by the visual fields (fig. 24). In the right eye, which showed the manifest atrophy, there was evidence of a cecocentral scotoma extending into the right field, in addition to the left hemianopia. Retests six years later showed a slight improvement in the hemianopic

defect in both eyes but an increase in the extent of the cecocentral scotoma in the right eye.

(d) *Patients with Active or Inactive Optic Neuritis.*—There were 23 patients in this group. The clinical findings are summarized in table

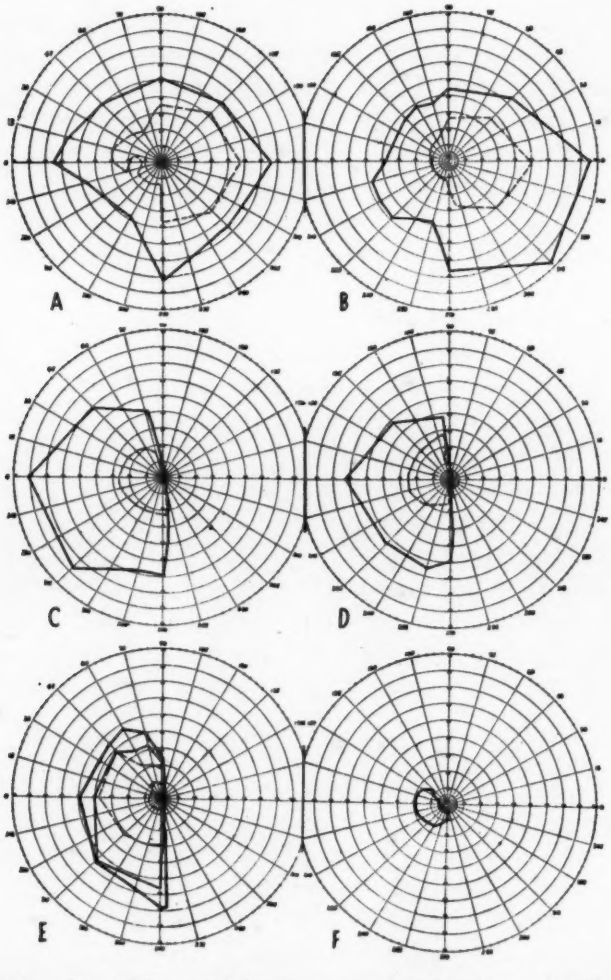

Fig. 22.—Complete and partial homonymous hemianopia. *A* and *B* (case 69) show the fields for the left and the right eye for 1 degree white (solid line) and 1 degree red (broken line) test objects. Vision for both eyes was 20/15. *C* and *D* (case 70) show the fields for the left and the right eye for 1 degree white (solid line) and 1 degree red (broken line) test objects. Vision for both eyes was 20/30. *E* and *F* (case 71) represent the left and the right visual field, the outer solid line in *E* bounding the field for a 3 degree white, the inner solid line that for a 1 degree white, the outer broken line that for a 1 degree red and the inner broken line that for a ½ degree red test object. The field in *F* is that for a 1 degree white test object. Vision for the left eye was 20/30 + 3 and for the right 10/200.

7. One patient had atrophy of the optic nerve secondary to old optic neuritis and 3 had active optic neuritis. Ophthalmoscopic examination of the other 19 patients showed deposition of glial tissue over the disk, blurred margins or other evidences of old low grade optic neuritis without actual secondary atrophy. The fields of the 3 patients tested during the active stage of optic neuritis are shown in figure 25.

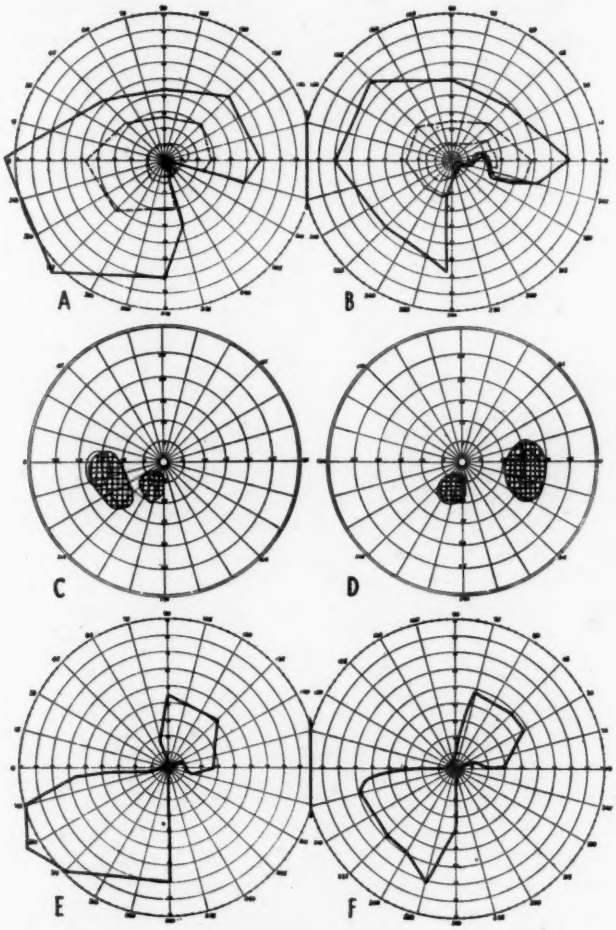

Fig. 23.—Homonymous quadrantic defects, homonymous scotomas and double quadrantic defects. *A* and *B* (case 72) show the left and the right visual field for 1 degree white (solid line), 1 degree red (outer broken line) and ½ degree red (inner broken line) test objects. Vision for both eyes was 20/15. *C* and *D* (case 73) represent the left and the right eye. Cross-hatched areas represent blindspot and scotoma for a 1 degree blue test object. Vision for both eyes was 20/15. *E* and *F* (case 75) show the left and the right visual field for a 3 degree white test object. Vision for both eyes was 20/40.

In case 81 the field defect consisted solely in an extensive, more or less concentric enlargement of the blindspot, which included the foveal area. The peripheral limits for 1 degree white and red test objects were entirely normal. Retests one year later showed only a slight enlargement of the blindspot and no foveal involvement. In case 82 the peripheral limits for a 1 degree white test object were entirely normal. In both eyes there were enlargement of the blindspot and paracentral scotoma, and in the left eye a large scotoma of the nerve fiber bundle type for a 1 degree blue test object. In case 83 the most marked defect was a nerve fiber bundle scotoma extending from the upper border of

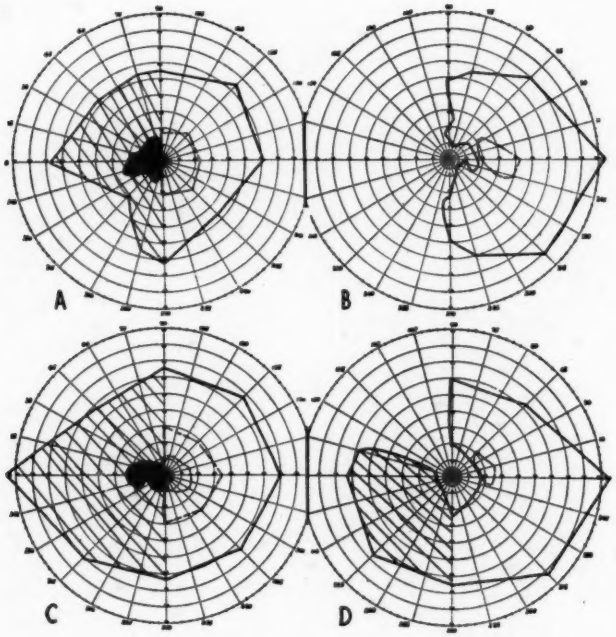

Fig. 24 (case 80).—Left homonymous hemianopia, associated with cecocentral scotoma of right eye. *A* and *B* show the left and the right visual field on May 12, 1931; *C* and *D* those on Jan. 20, 1937. The solid line bounds the field for a 1 degree white test object, the long dashes that for 1 degree red and the short dashes that for a ½ degree red test object. The solid area represents absolute scotoma and the area covered by slanting lines relative scotoma for a 1 degree white test object. Vision in the left eye was 20/20 at the first examination and 20/30 + 3 at the second; that in the right eye was reduced to perception of hand movements at both examinations.

the blindspot. Its location corresponded to the ophthalmoscopic finding of blurring and edema at the lower nasal border of the disk.

The patient whose disks showed definite secondary atrophy (case 84) had advanced field defects consisting of cecocentral scotomas and

Case No. and Date Examined	Observations in Visual Fields	Results of Ophthalmoscopic Examination	Corrected Visual Acuity	Type of Syphilis	Comment
69 4/14/36	Partial left homonymous hemianopia (fig. 22)	O. D. Normal O. S. Questionable temporal pallor of nerve head	O. D. 20/15 O. S. 20/15	Diffuse meningovascular	Left hemiplegia in 1931
70 1/15/35	Complete right homonymous hemianopia (fig. 22)	O. U. Nerve heads normal; moderate retinal arteriosclerosis	O. D. 20/30 O. S. 20/30	Diffuse meningovascular	History of transient paraplegia at onset of hemianopia in March 1934
71	Right homonymous hemianopia with some involvement of left side, especially in field of right eye (fig. 22)	O. D. Questionable temporal pallor of nerve head O. S. Normal	O. D. 10/200 O. S. 20/30+3	Diffuse meningovascular	Neurologic evidence of bilateral central lesions involving left temporal lobe and right central hemisphere (aphasia, left facial palsy and left hemianesthesia); diabetes mellitus
72	Lower right homonymous quadrantic defects (fig. 23)	O. U. Nerve heads normal; retinal arteriosclerosis	O. D. 20/15 O. S. 20/15	Latent; no evidence of involvement of central nervous system	History of transient blindness, advanced hypertension and cerebral arteriosclerosis
73 2/6/33	Homonymous scotoma in lower left and right field (fig. 23)	O. U. Questionable pallor of nerve heads	O. D. 20/15 O. S. 20/15	Dementia paralytica	Sudden attack of complete blindness and aphasia in 1932; vision returned in 3 days on right side; syphilitic aortitis
74	Right homonymous hemanopia	O. D. Questionable pallor of nerve head; some connective tissue over cup O. S. Normal	D. D. 20/30 O. S. 20/200 (corneal opacity)	Tertiary cardiovascular	Aortitis; aortic insufficiency; defects in memory; partial anesthesia of right side and weakness of right leg; central arteriosclerosis; question of multiple sclerosis
75	Upper left and lower right quadrantic defects (fig. 23)	O. U. Nerve heads normal; a little macular degeneration; some arteriosclerosis	O. D. 20/40 O. S. 20/40	Latent; no evidence of involvement of central nervous system	History of repeated cranial traumatism and cerebral injury; transient attacks of blindness; marked hypertension and arteriosclerosis
76	Upper left homonymous quadrantic defects	O. U. Cupping a little blurred; nerve heads otherwise normal	O. D. 20/30—1 O. S. 20/50+ O. S. has high refractive error (—8.00 sph. and —3.50 cyl.)	Dementia paralytica	History of preceding cranial injury from a fall; roentgenogram of skull shows areas of calcification and questionable blood clot
77	Lower right homonymous quadrantic defects	O. U. Questionable pallor of nerve heads; retinal arteriosclerosis	O. D. 20/15—2 O. S. 20/30+3	Asymptomatic late tertiary cardiovascular	Violent injury to head in 1918; roentgenogram shows defects in frontal bone; large irregular sella; chronic aortic insufficiency
78 12/2/31	Upper left homonymous quadrantic defects	O. U. Normal	O. D. 20/15 O. S. 20/15—1	Diffuse meningovascular	History of partial paraplegia in 1930; chronic anterior poliomyelitis
79	Lower right homonymous quadrantic defects	Nerve heads hyperemic	O. D. 20/15 O. S. 20/15	Tabes	No history of cerebral injury, paralysis or other involvement of central nervous system
80 5/12/31	..... Left homonymous hemianopia, with cecocentral scotoma in right eye	..... O. D. Advanced primary atrophy of optic nerve O.S. Questionable primary atrophy of optic nerve; mild retinal arteriosclerosis	O. D. Hand motion O. S. 20/20	Dementia paralytica	No history of cerebral injury, paralysis or other involvement of central nervous system; mild general arteriosclerosis
1/20/37	O. U. Partial recovery on left side O. D. Increase in extent of cecocentral scotoma O. S. Questionable early defect at fixation point for ½° red (fig. 24)	O. D. Advanced primary atrophy of optic nerve O. S. Early primary atrophy of optic nerve	O. D. Hand motion O. S. 20/30+3		

TABLE 7.—*Patients with Active or Inactive Optic Neuritis*

Case No. and Date Examined	Observations in Visual Fields	Corrected Visual Acuity	Type of Syphilis	Results of Ophthalmoscopic Examination
81 6/24/35	O. D. Marked concentric enlargement of blindspot with relative scotoma extending to fovea O. S. Not tested (amblyopia exanopsia)	O. D. 20/100+1	Of central nervous system; early neurorecurrence	O. D. Neuroretinal margins blurred; disks congested and elevated 2 diopters; exudates extending into surrounding retina; a few small flamelike hemorrhages present
6/29/36*	O. D. Slight enlargement of blindspot; fovea not involved (fig. 25)	O. D. 20/20+2	.....	O. D. Edges of disk only slightly blurred; disk grayish
82† 2/9/34	O. D. Scotoma below fovea, not connected with blindspot O. S. Scotoma in lower nasal quadrant, and nerve fiber bundle scotoma extending from upper border of blindspot	O. D. 20/40— O. S. 20/30	Early secondary, cutaneous, with alopecia	
2/16/34	O. U. Slight improvement (fig. 25)	O. D. 20/40 O. S. 20/20—2	.....	O. U. Blurred margins and obliterated cups; no measurable elevation in O. D., about 1 diopter in O. S.
83	O. D. Not tested (amblyopic) O. S. Nerve fiber bundle defect extending from upper temporal border of blindspot; general peripheral contraction (fig. 25)	O. S. 20/40	Of central nervous system; early neurorecurrence	O. S. Slight pallor of disk; definite edema in lower nasal quadrant, with some edema of adjacent retinal fibers
84	O. U. Cecocentral scotomas and marked peripheral contraction for 1° white (fig. 26)	O. D. 3/200 O. S. 3/200	Dementia paralytica	O. U. Secondary atrophy of optic nerves; disks flat and slightly pale; glial tissue in cups and along vessels a short way out from disks; peripheral fundi normal
85	O. U. Definite peripheral contraction, especially marked in upper field for 1° white; slight defects in upper field of each eye for 1° red, connected with blindspot in left (fig. 27)	O. D. 20/15 O. S. 20/15	Dementia paralytica	Disks show blurred neuroretinal margins and blurred cups; no activity
86	O. U. Marked peripheral contraction for 1° white; slight contraction for 1° red; slight enlargement of blindspot (fig. 27)	O. D. 20/15—2 O. S. 20/15—2	Tabes	Disks show a little blurring of neuroretinal outlines and definite increase in capillarity
87	O. U. Concentric peripheral contraction for 1° white and color; enlargement of blindspots (fig. 27)	O. D. 20/15—1 O. S. 20/15	Dementia paralytica	Disks flat and slightly irregular; some perivascular connective tissue on vessels over disk; no activity
88	O. U. Concentric contraction, marked for 1° white, slight for 1° blue and red; slight enlargement of blindspot in right eye	O. D. 20/15 O. S. 20/20—3	Latent; no evidence of involvement of central nervous system	O. U. Slight hyperemia of disks and blurred cups
89	O. U. Peripheral contraction, definite for 1° white, questionable for 1° color; blindspots normal	O. D. 20/15 O. S. 20/15—1	Tabes	A little fibrosis over cups; possibly slight pallor
90	O. D. Slight contraction for 1° white; questionable contraction for 1° red; blindspot for ½° white enlarged O. S. Slight contraction for 1° white and red; normal blindspot for ½° white (fig. 27)	O. D. 20/15 O. S. 20/15	Dementia paralytica	O. D. Nerve head a little blurred; cup lost O. S. Nerve head almost normal; cup lost

* Metamorphopsia in region 5° to right of fixation point.
† Patient notices constant "flickering" before eyes.

TABLE 7.—*Patients with Active or Inactive Optic Neuritis*—Continued

Case No. and Date Examined	Observations in Visual Fields	Corrected Visual Acuity	Type of Syphilis	Results of Ophthalmoscopic Examination
91‡ 6/8/32	O. U. Concentric contraction for 1° white; normal fields for 1° blue and red O. S. Slight enlargement of blindspot	O. D. 20/15—3 O. S. 20/15	Tabes	O. U. Disks good color; cups obliterated; neuroretinal margins a little blurred
10/26/32	O. U. Fields normal	O. D. 20/15 O. S. 20/15	.....	O. U. Disks good color; right cup obliterated and left just seen; a little connective tissue along vessels
92	O. U. Fields for 1° white normal and for 1° colors show questionable contraction O. D. Blindspot enlarged O. S. Normal blindspot (fig. 28)	O. D. 20/15 O. S. 20/15	Early secondary, cutaneous	O. D. Neuroretinal margins a little blurred; connective tissue in cup; no atrophy O. S. Slight blurring of cup
93	O. D. Blindspot for ½° blue and ½° red shows scotomatous extension; fields otherwise normal (fig. 28) O. S. Normal	O. D. 20/15 O. S. 20/15	Diffuse meningovascular	O. D. Nerve head shows blurred margins, blurred cup, a little connective tissue; is possibly slightly paler than left O. S. Blurred cup
94	O. D. Normal peripheral fields for 1° white and color; questionable enlargement of blindspot O. S. Questionable contraction of the fields for 1° white and color; questionable enlargement of blindspot	O. D. 20/15—1 O. S. 20/15—1	Early secondary, recurrent, cutaneous and arsphenamine resistant	O. U. Nerve heads show blurred margins; cups obliterated; connective tissue formation along vessels; no activity
95	O. D. Normal O. S. Field for 1° white shows slight contraction; field for 1° blue and red, normal; blindspot normal	O. D. 20/15—1 O. S. 20/15—2	Diffuse meningovascular and tertiary osseous, with osteitis of skull	O. U. Disks flat, of good color; a little fibrosis over cups
96	O. D. Normal O. S. Normal except for questionable defect in field for 1° red	O. D. 20/15 O. S. 20/15	Tabes	O. U. Disks flat, of good color; a little fibrosis over cups
97	O. D. Normal O. S. Questionable contraction of fields for 1° white, blue and red; blindspot normal	O. D. 20/15—3 O. S. 20/15—2	Dementia paralytica	O. U. Fundi a little hyperemic and cups indistinct; otherwise normal
98	O. U. Field for 1° white normal; slight contraction for 1° red; questionable enlargement of blindspot	O. D. 20/15—2 O. S. 20/15—2	Dementia paralytica; at autopsy small hypophysial adenoma	O. U. Glial tissue formation over disks
99	O. D. Normal O. S. Scotomatous enlargement of blindspot for ½° color; otherwise normal	O. D. 20/15—3 O. S. 20/15—3	Dementia paralytica	O. U. Disks flat, of good color; cupping a little indistinct
100	O. U. Normal	O. D. 20/15—2 O. S. 20/15—2	Asymptomatic of central nervous system	O. U. Disks flat, of good color; cupping blurred
101	O. U. Normal	O. D. 20/15 O. S. 20/15—1	Dementia paralytica	O. U. Disks flat, of good color; cupping slightly blurred
102	O. U. Normal	O. D. 20/20 O. S. 20/20	Diffuse meningovascular	O. U. Disks flat, of good color; cupping slightly blurred
103	O. U. Normal	O. D. 20/20 O. S. 20/15	Dementia paralytica	O. U. Disks a little vascular; cups blurred but of good color

‡. Subarachnoid hemorrhages and choked disk in January 1932.

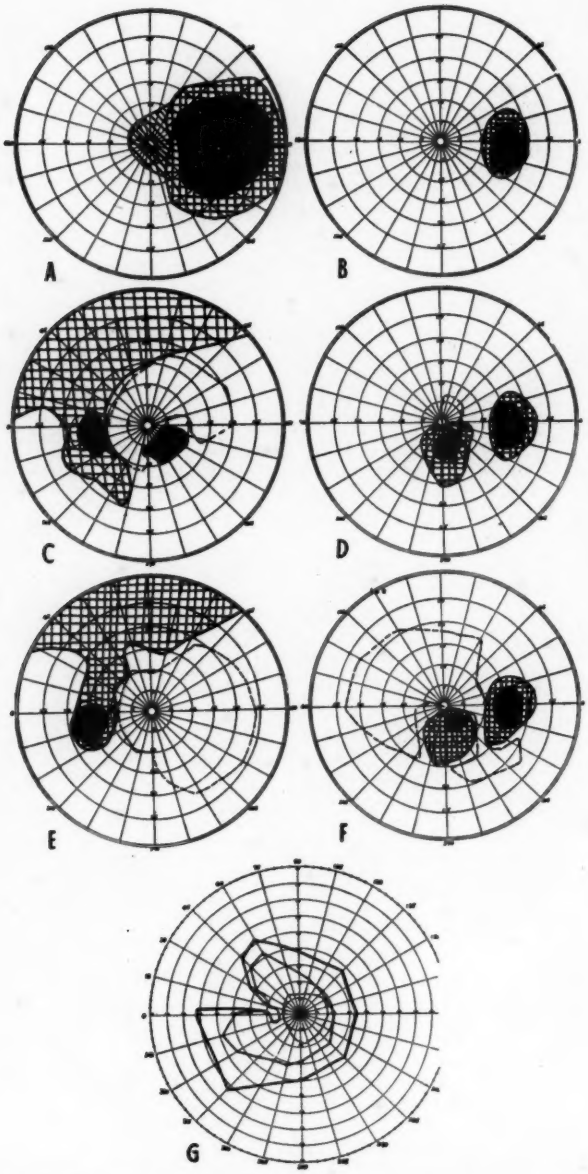

Fig. 25.—Visual fields in cases of active optic neuritis. *A* and *B* (case 81) represent the right eye on June 24, 1935, and June 29, 1936, respectively. In *A*, the solid area represents the blindspot for a 1 degree white test object; the cross-hatched area, that for a 1 degree red object and the area covered by slanting lines, relative scotoma for 1 degree white and red objects. Vision was 20/50. In *B* the solid area represents the blindspot for a ½ degree white test object and the cross-hatched area that for a ½ degree red test object. Vision was 20/20 + 2. The peripheral field for white and colored test objects was normal at both determinations. *C, D, E* and *F* (case 82) represent the fields of the left and the right eye on Feb. 9 and on Feb. 16, 1934. A solid area represents blindspot or scotoma for a ½ degree white test object and a cross-hatched area blindspot or scotoma for a 1 degree blue test object. The broken line represents the boundary of the field for a 1 degree blue test object. At the first determination vision for the left eye was 20/30 and for the right 20/40—. At the second determination vision for the left eye was 20/20 —2 and for the right 20/40. The peripheral field for a 1 degree white test object was normal in both case 81 and case 82. *G* (case 83) shows the left visual field for 3 degree white (outer solid line), 1 degree white (inner solid line) and 1 degree blue (broken line) test objects. Vision was 20/40.

marked peripheral contraction for a 1 degree white test object. The visual acuity of each eye was reduced to 3/200 (fig. 26).

The 19 patients who had changes in the fundi indicative of previous low grade optic neuritis which was inactive at the time of examination

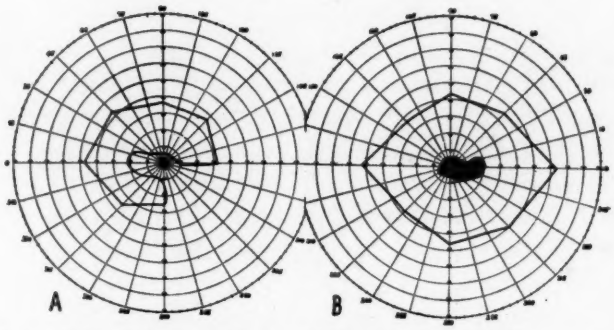

Fig. 26 (case 84).—Visual field *A* of left and *B* of right eye for a 1 degree white test object in a case of secondary atrophy of the optic nerve. Vision was 3/200 for both eyes. The solid area represents scotoma for a 1 degree white test object.

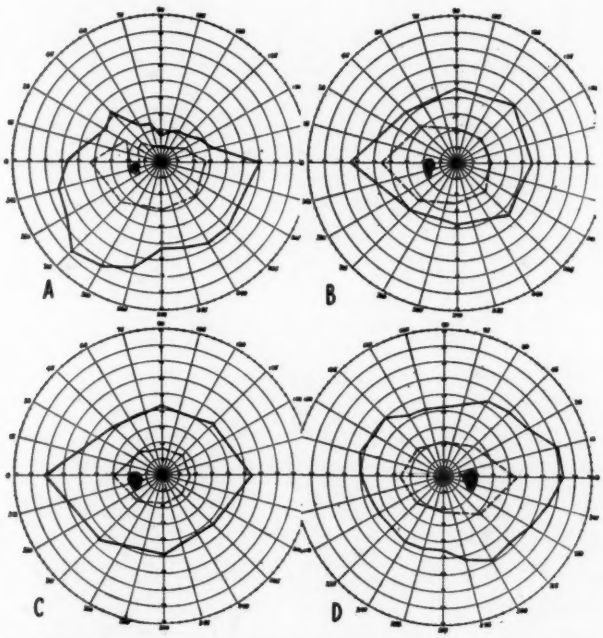

Fig. 27.—Slight defects in the fields of patients with inactive low grade optic neuritis. *A* (case 85), *B* (case 86) and *C* (case 87) show the left visual field and *D* (case 90) the right visual field for 1 degree white (solid line) and 1 degree red (broken line) test objects. The solid area in each illustration represents the blindspot for a ½ degree white test object. In *A*, *C* and *D*, vision was 20/15; in *B*, 20/15 — 2.

had visual acuity of 20/20 or better. The perimetric findings included slight but definite field defects, questionable defects and definitely normal fields. Seven patients had definite defects, such as are illustrated in figure 27. These consisted in localized or concentric contraction of the peripheral limits for white, for colored or for both white and colored test objects and slight defects connected with the blindspot, giving either concentric enlargement or nerve fiber defects extending from the upper or the lower border. No other types of scotoma were found. The fields of 8 patients showed only questionable deviations from normal, either in the peripheral limits for white or colored test objects or in the size of the blindspot. (Such borderline defects are illustrated in figure 28.) Four patients had definitely normal fields for 1 degree white, blue and red test objects and normal blindspots for ½ degree white, blue and red objects.

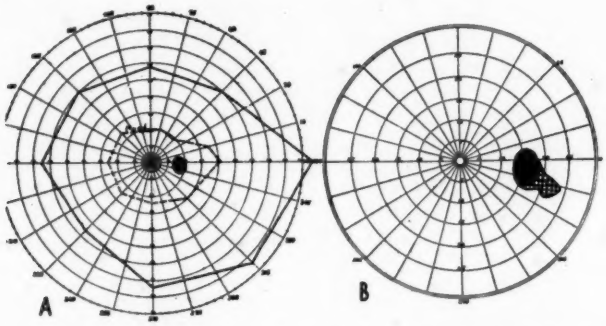

Fig. 28.—Questionable defects in the fields of patients with inactive low grade optic neuritis. *A* (case 92) shows the right visual field for 1 degree red (broken line) and 1 degree white (solid line) test objects. The solid area represents a blindspot for a ½ degree white test object. Vision was 20/15. In *B* (case 93) the solid area represents a blindspot for a ½ degree white test object and the cross-hatched area one for a ½ degree blue object in the right field of a patient whose peripheral fields for white and colored test objects were normal. Vision was 20/15.

Comment.—The fields of the 3 patients with active optic neuritis showed, variously, enlargement of the blindspot, central and paracentral scotomas and nerve bundle defects. The patient who had definite secondary atrophy after the neuritis subsided showed concentric contraction and nerve bundle defects with scotomas, while the 19 patients who recovered without secondary atrophy had normal vision and either normal fields or only slight defects.

On the basis of this knowledge it appears that the initial field change in cases of optic neuritis may be only an enlargement of the blindspot. If the inflammation of the optic nerve is sufficiently severe, there may be nerve bundle defects and central or paracentral scotoma,

and the changes may resemble those in cases of primary atrophy of the optic nerve. In patients with severe neuritis, secondary atrophy of the nerve may ensue and these changes in the field become permanent. However, in the majority of the patients the inflammation in the nerve subsides and leaves no serious sequelae; vision is normal and the visual fields either are normal or have defects limited to enlargement of the blindspot and slight changes for white or color. Enlargement of the blindspot and a normal field for white associated with a concentrically contracted field for color were not found in cases of primary atrophy of the optic nerve and appear characteristic of old optic neuritis.

GENERAL COMMENT

In the light of these studies on syphilitic patients with primary atrophy of the optic nerve, with normal fundi and field defects, with homonymous field defects and with evidences of active or inactive optic neuritis, there are certain obvious general conclusions.

It appears that the conclusions of Uhthoff, which have dominated the thought of ophthalmologists on field changes incident to primary syphilitic atrophy of the optic nerve for over thirty years, are incorrect. Central or cecocentral scotoma, far from being uncommon, occurs in over 50 per cent of cases. The view that such scotoma is the result of an involvement of the exposed optic tracts at the base of the brain by a basilar syphilitic meningitis also appears to be in error. Such central scotoma has been observed with equal frequency in patients with parenchymatous neurosyphilis and in patients with diffuse meningovascular syphilis.

It is obviously preferable to demonstrate the position of a given lesion from histologic evidence rather than to postulate its position from clinical evidence. Nevertheless, in the absence of histologic material, the clinical evidence here presented appears to give definite information relative to the location in the optic neuro-pathways of the lesion responsible for the field defects.

The absence of homonymous defects in the fields in any of the 56 patients with frank primary syphilitic atrophy of the optic nerve makes untenable the view that the primary lesion responsible for the atrophy is posterior to the chiasm. Likewise, the finding of cerebral trauma or of arteriosclerosis with vascular accident in 84 per cent of the syphilitic patients with homonymous defects in the fields is evidence that when homonymous defects are present in syphilitic patients they are probably due to those causes rather than to degenerative syphilitic lesions involving the tracts, the geniculate bodies or the optic radiations. Therefore, the primary lesion responsible for such a defect must lie in the chiasm or in the optic nerve.

When first considered, the tendency toward similar or heteronymous defects in the two eyes would appear to be evidence in favor of a chiasmal lesion. Otherwise it would be necessary to assume lesions occupying similar positions in the two nerves to explain this peculiar and characteristic finding. The assumption of a lesion in the periphery of the chiasm might account for such symmetric changes as altitudinal hemianopia and bitemporal defects. A widespread involvement of the entire periphery of the chiasm might account for concentrically contracted fields in both eyes. However, the heteronymous defects in the fields in cases of syphilitic primary atrophy of the optic nerve do not in general show any resemblance to changes in the visual fields produced by other known chiasmal lesions. Binasal defects associated with normal temporal fields, as in case 9 (fig. 4), could be produced only by two separate chiasmal lesions, and type 3 defects (bilateral cecocentral scotomas and normal peripheral fields) are likewise difficult to explain on the basis of a chiasmal lesion. Fields such as those in case 48 (fig. 13), with definite nasal steps, could not be produced by a lesion at the chiasm. Consequently, aside from the fact that such a location does not explain bilateral symmetry in the fields, the findings argue for lesions located in the optic nerves rather than in the chiasm.

If the view that the fibers supplying the outer regions of the visual field have a peripheral location in the nerve is accepted, the various forms of field defect can be explained on the basis of a lesion starting somewhere in the periphery of the optic nerve. A diffuse lesion affecting the entire circumference of the nerve would produce concentric peripheral contraction of the visual field. If the lesion were in the foraminal or the intracranial portion of the nerve, the cecocentral area of the field would be involved late, owing to the axial location of the papillomacular bundle. A defect of type 1 would consequently be expected as a result of a lesion in the posterior portion of the nerve. If, however, the lesion were located near the papilla, where the papillomacular bundle has a peripheral location, a diffuse perineural lesion would produce a cecocentral scotoma in addition to the peripheral contraction. Isolated cecocentral scotoma, nasal steps and combinations of these two defects would result from a lesion in the anterior portion of the nerve involving the papillomacular bundle, the fibers contiguous to it or both. Other localized defects in the visual fields would result from a more or less circumscribed lesion in either the anterior or the posterior part of the optic nerve.

It is difficult to explain the peculiar and almost characteristic similarity of the defects in the fields of the two eyes in cases of primary atrophy. As already pointed out, altitudinal and bitemporal defects may possibly be due to a lesion starting in the chiasm, but to explain

the great majority of these defects it appears necessary to assume that similar lesions are present in the two optic nerves.

The observation that field defects for colored and for small white objects may either regress or completely disappear argues in favor of Stargardt's conclusion that the primary lesion in cases of syphilitic atrophy of the optic nerve is a peripheral and interstitial neuritis and that the actual degeneration of the nerve fibers is secondary to this inflammatory change. If antisyphilitic therapy can be instituted before the nerve fibers are damaged beyond repair, there is reasonable hope that the peripheral and interstitial neuritis may be controlled and vision and field defects clear without permanent damage. This is in line with Moore's contention that antisyphilitic therapy in cases of atrophy of the optic nerve offers hope only when instituted before vision has fallen to any great degree.

SUMMARY

The field defects in a group of 56 patients with primary syphilitic atrophy of the optic nerve were of four separate types: (1) concentric contraction of the peripheral field associated with late loss of vision; (2) sector-shaped, or nerve bundle, defects, with which loss of vision might be early or late, depending on the involvement of the papillomacular bundle; (3) central or cecocentral scotoma with normal peripheral fields, associated with early loss of visual acuity and (4) central or cecocentral scotoma with defects in the peripheral fields, also associated with early loss of vision.

Central or cecocentral scotoma was found in 53 per cent of cases of primary atrophy of the optic nerve.

A separate group of neurosyphilitic patients showed defects in the visual fields and normal optic nerves. A study of these patients and of patients with frank syphilitic atrophy of the optic nerve indicates that perimetric changes may antedate either visual failure or pallor of the disk in the development of syphilitic atrophy of the optic nerve.

Homonymous defects in the visual fields when present in patients with neurosyphilis appear to be dependent on such factors as cerebral trauma and arteriosclerosis with vascular accident, rather than on syphilitic involvement of the optic tracts or radiations.

The defects in the visual fields in cases of syphilitic optic neuritis may simulate the changes in cases of primary syphilitic atrophy. There is usually a tendency to almost complete recovery, with no residuum other than enlargement of the blindspot or a minor peripheral defect.

The clinical evidence from these studies indicates that the site of the lesion responsible for syphilitic atrophy of the optic nerve and visual field defects lies in the optic nerve rather than in the chiasm or posterior to it and is probably a peripheral and interstitial neuritis associated with secondary degeneration of the nerve fibers.

CANCER OF THE EYELIDS, CONJUNCTIVA AND CORNEA

II. SQUAMOUS CELL EPITHELIOMA

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The purpose of this report is to emphasize the importance of squamous cell epithelioma of the eyelids, conjunctiva and cornea. The material is taken from a series of 464 cases [1] of carcinoma of all types which involved the eyelids and the conjunctiva. A few cases of squamous cell epithelioma of the cornea are included.

Histologic examination was performed in all cases except those in which treatment consisted entirely of radium therapy and in which, consequently, biopsy was not performed. As the basis for the histologic portion of the study, 230 cases were chosen. In 59, or approximately 25 per cent of the histologic material, the growths were squamous cell epitheliomas. Basal cell epithelioma and mixed basal cell and squamous cell epithelioma composed 75 per cent of the 230 tumors and have been considered elsewhere.[2]

OCCURRENCE

Squamous cell epithelioma may involve any portion of the eyelids, conjunctiva or cornea. The area most frequently involved is the lower eyelid (fig. 1). All types of epithelioma occur more often on the lower lid than elsewhere about the eye, but squamous cell lesions occur less frequently on the lower eyelid than do any other types of epithelioma (fig. 1).

The distribution of squamous cell lesions about the eye differs from that of other types of epithelioma in that the conjunctival surfaces are

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1. Birge, H. L.: Epithelioma of the Eyelids, Conjunctiva and Cornea, thesis submitted to the faculty of the Graduate School of the University of Minnesota, 1937.

2. Birge, H. L.: Cancer of the Eyelids: I. Basal Cell and Mixed Basal Cell and Squamous Cell Epithelioma, Arch. Ophth. **19**:700-708 (May) 1938..

so frequently involved (fig. 1). Approximately 37 per cent of the squamous cell lesions in this series were removed from the conjunctiva, the cornea or the junction of the conjunctiva and the cornea, the limbus.

In approximately 20 per cent of the cases of squamous cell epithelioma in which the histologic grade of malignancy was determined the lesion occurred on the conjunctival surface of a lid or the globe. The corneal limbus was involved in slightly more than 10 per cent of the cases. The cornea alone was involved by squamous cell lesions in approximately 5 per cent of this group. Primary corneal malignant growths are rare, and cases of this sort will be dealt with in detail subsequently.

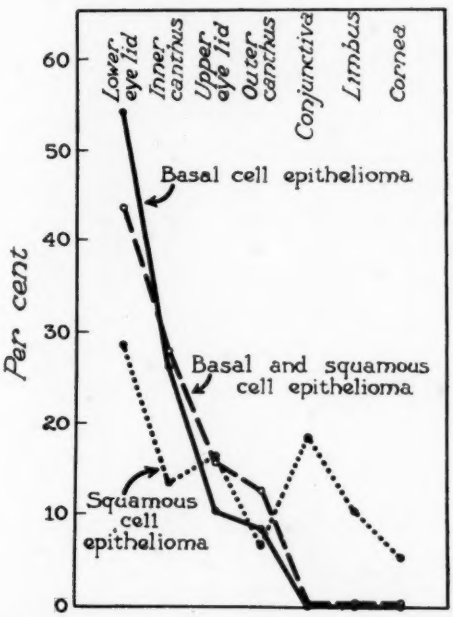

Fig. 1.—Chart showing the relative frequency of the situation of the various types of epithelioma of the eyelids and adnexa in a series of 230 cases.

HISTOLOGIC GRADE OF MALIGNANCY

The pathologic material was graded according to Broders' method (table 1). In this series squamous cell lesions of grades 2 and 3 occurred most frequently. The most highly malignant lesions, those of grade 4. were least common.

ETIOLOGY

Squamous cell epithelioma is known to occur frequently at the junction of two different types of epithelium.[3] There are two places about

3. Benedict, W. L., and Knight-Asbury, M.: The Treatment of Malignant Lesions of the Eyelids, New York State J. Med. **29**:675-677 (June 1) 1929.

the eye where the epithelium undergoes a change in type: One is at the junction of the conjunctiva and the skin of the eyelid; the other, at the junction of the conjunctiva and the cornea.

A factor of considerable importance in the causation of epitheliomas in general is chronic irritation. In this group the squamous cell lesions were associated with a history of chronic irritation and trauma in nearly the same percentage of cases as were other types of epithelioma reviewed in the entire series.[1] Of the patients with squamous cell epithelioma, 32.2 per cent gave a history which supported the belief that the lesion arose as a result of chronic irritation or trauma. Among the factors listed as being associated with the development of a squamous cell lesion were warts, moles and chalazions. Some patients stated that squeezing a pimple on a lid resulted in the growth. Others stated

TABLE 1.—*Grade and Situation About the Eye of Fifty-Nine Squamous Cell Epitheliomas*

Grade	Conjunctiva	Cornea	Limbus	Outer Canthus	Inner Canthus	Upper Lid	Lower Lid	Total
1	1	..	2	..	1	1	3	8
2	6	1	4	..	3	3	4	21
3	1	2	..	4	2	3	6	18
4	2	..	..	..	..	2	2	6
Not graded	1	..	..	..	2	1	2	6
Total in various situations	11	3	6	4	8	10	17	59

that being hit in the eye with a cow's tail or getting the lid scratched by a thorn or a finger nail led to the occurrence of the growth. One man was struck by lightning, and shortly thereafter a tumor of the eyelids developed. A few felt that pressure of long standing exerted by glasses on the side of the nose caused the lesion. Chronic blepharitis or meibomitis of long standing was associated with squamous cell epithelioma of the eyelids in 1 case.

Correlation did not occur between the type of chronic irritation and the type or grade of epithelioma. Most of the men were farmers and were exposed only to the elements. Lane [4] and Kennaway [5] have dealt with the occurrence of malignant lesions of the lids in workers exposed to carcinogenic agents, such as pitch and tar. Apparently cancer of the eyelids develops less frequently in workers who come in contact with carcinogenic agents than in persons exposed to the ordinary types of

4. Lane, L. A.: An Occupational Study of Cancer of the Eye and Adnexa, Surg., Gynec. & Obst. **64**:458-464 (Feb. 15) 1937.

5. Kennaway, E. L.: The Anatomical Distribution of the Occupational Cancers, J. Indust. Hyg. **7**:69-93 (Feb.) 1925.

chronic irritation and trauma of everyday life. This should not minimize the importance of tar and allied chemicals as carcinogenic agents but merely shows that in a group of patients in whom malignant growths of the eye and eyelids developed many other types of irritation were present.

Inadequate treatment of a benign lesion of the eyelids may have a bearing on the development of a malignant growth. Many of the patients in this series had been treated with various salves, roentgen irradiation and the electric needle before the growths were excised. Treatment that does not completely destroy a lesion produces a reaction of inflammation in the lesion which may be a carcinogenic factor if continued long enough. Chalazions which have become malignant under treatment have been reported.[6] It does no harm to take specimens for biopsy from recurrent chalazions. Sometimes the first sign of recurrence of such tumors is metastasis to the parotid or submaxillary glands.[7]

Age is an etiologic factor in squamous cell epithelioma, as it is in all types of malignant growths. Most of the squamous cell lesions in this series occurred in the fifth decade of life, one decade earlier than that in which the basal cell lesions occurred.[2] The youngest person in this series to have a squamous cell epithelioma was a girl 3½ years of age. None of the other types of epithelioma occurred at as young an age.

Sex also may be an etiologic factor in squamous cell lesions. Of the patients who had such lesions, 76 per cent were men.

TREATMENT

Treatment in the 53 cases of squamous cell epithelioma reviewed here in which the malignancy of the growths was graded was about equally divided between surgical operation alone and operation in combination with irradiation (table 2). None of the cases in which treatment consisted of irradiation alone is considered. Treatment of lesions of the eyelids by irradiation alone usually means that treatment is given without knowledge of the pathologic diagnosis.

The surgical measures employed were excision, cauterization, fulguration and plastic repair, as indicated. In 1 case only was simple

6. DéJean, C.; Harant, and Vernières: Pseudo-tumeur récidivant de la paupière à cellules géantes sarcoplasmiques, Arch. Soc. d. sc. méd. et biol. de Montpellier **15**:240-244 (May) 1934; abstracted, Am. J. Cancer **24**:167 (May) 1935. Kiewe, P.: Ueber eine rezidivierende, unter dem Bilde eines Chalazions auftretende Geschwulst der Lider (Zur Frage eines entzündlichen Pseudotumors der Lider), Acta ophth. **13**:139-149, 1935.

7. Hagedoorn, A.: Adenocarcinoma of a Meibomian Gland, Arch. Ophth. **12**:850-867 (Dec.) 1934.

enucleation required; that was a case of a malignant lesion of the limbus, grade 2. Exenteration of the contents of the orbit was required with increasing frequency as the histologic grade of malignancy increased (table 2). Exenteration was performed in 25 per cent more cases in which the lesions were of grade 3 than in cases in which they were of grade 2, and exenteration was performed in 60 per cent more cases in which the lesions were of grade 4 than in cases in which they were of grade 3.

RESULTS

The malignancy of squamous cell epitheliomas as a group, judged by the percentage of mortality, is greater than that of any or of all other

TABLE 2.—*Behavior of Squamous Cell Epitheliomas on the Basis of Histologic Grading*

	No. of Patients		Died of Ocular Cancer		Recurrences		Invasion of Orbit		Treatment, No. of Patients		Blind in Affected Eye		Exenteration	
Grade	Total	Traced	No. of Patients	Percentage	No. of Patients	Percentage	No. of Patients	Percentage	Surgical Treatment Alone	Surgical Treatment and Irradiation	No. of Patients	Percentage	No. of Patients	Percentage
1	8	6	0	0	0	0	0	0	5	3	0	0	0	0
2	21	20	3	15.0	10	50.0	6	30.0	8	13	5	25.0	3	15.0
3	18	15	7	46.7	14	93.3	9	60.0	9	9	8	53.3	6	40.0
4	6	5	4	80.0	3	60.0	5	100.0	4	2	5	100.0	5	100.0
Ungraded growths	6													

types of epithelioma (except melanotic epithelioma) found on the eye or the adnexa. The correlation between the clinical behavior and the grade of histologic malignancy was close in the series reviewed here. All squamous cell lesions are not of equal malignancy. A follow-up study of patients, in some instances for more than fifteen years, demonstrated that the histologic grading of the malignancy bears a close relation to the clinical course over a period of years.

Squamous cell lesions of grade 1 of the eye or eyelids did not cause any deaths in this series, or the loss of any eyes. There were no recurrences after treatment, nor was there invasion into the orbit. Cure was effected in all cases by surgical excision (table 2).

Squamous cell lesions of grade 2 were the most frequent type of lesion in this series (table 1). They caused loss of the affected eye in 25 per cent of the cases and were responsible for the death of 15 per cent of the persons affected. Recurrences were frequent, and invasion of the orbit occurred in 30 per cent of the cases (table 2).

Squamous cell lesions of grade 3 were next in frequency of occurrence (table 2). They caused loss of the affected eye in 53 per cent of the cases and death in 46 per cent. There were recurrences in nearly all cases following all types of treatment. This includes cases in which treatment consisted of inacceptable remedies as well as those in which treatment was surgical. Apparently, satisfactory initial treatment of lesions of grade 3 must be more vigorous. In 60 per cent of these cases the growth invaded the orbit. In 40 per cent, exenteration of the contents of the orbit was required.

Squamous cell lesions of grade 4 of the eye or adnexa occurred with least frequency of all grades of epithelioma (table 1); yet this type of lesion accounted for more deaths than did lesions of grade 2. There was invasion of the orbit in every case, followed by exenteration, and in 80 per cent of the cases the lesion proved fatal (table 2).

The behavior of lesions of grade 4 in no way resembled that of lesions of grade 1 (fig. 2). None of the persons with lesions of grade 4 survived five years, although 75 per cent were living three years after treatment at the clinic (table 3). Persons with lesions of grade 3 demonstrated the same percentage of survival after three years as did those who had lesions of grade 4. After the first ten years, 100 per cent of persons who had lesions of grade 3 were free from the effects of carcinoma. This fact may be due to the small number of cases, as it probably is, but it supports the idea that if a person who has a malignant lesion survives ten years his chances of dying of cancer are materially reduced.

When the period of survival of persons who have lesions of grade 2 is considered, it is seen that a relatively high percentage survived five years or more (table 3), but that during the next five years many persons succumbed to the effects of the epithelioma. The majority of patients in this group died in the second five years. None of the patients who had squamous cell growths of grade 2 lived fifteen years; those with malignant growths of grade 3 proved an exception (fig. 2).

While the survival periods of persons with lesions of various grades of malignancy yield some interesting figures, a more graphic picture of the relative malignancy of the various grades is obtained from figure 3. This graph is based on the data in table 2 and, in addition, shows the malignancy of the other types of epithelioma, namely, basal cell epithelioma and mixed basal cell and squamous cell epithelioma of the eye or eyelids. It is easy to see that (fig. 3) although squamous cell epithelioma may be more malignant than all other types of epithelioma about the eye (except melanotic epithelioma) this evidence of greater malignancy is due to the behavior of growths of grades 3 and 4.

TABLE 3.—*Survival Period of Patients with Squamous Cell Epithelioma on the Basis of Histologic Grading*

Grade	Total Cases	No. of Patients Operated On*	No. of Patients Traced	Lived 3 or More Years After Operation				Lived 5 or More Years After Operation				Lived 10 or More Years After Operation				Lived 15 or More Years After Operation	
				No. of Patients	Percentage of Traced Patients	No. of Patients Operated On*	No. of Patients Traced	No. of Patients	Percentage of Traced Patients	No. of Patients Operated On*	No. of Patients Traced	No. of Patients	Percentage of Traced Patients	No. of Patients Operated On*	No. of Patients Traced	No. of Patients	Percentage of Traced Patients
1	8	7	6	6	100	5	4	4	100	1	1	1	100	0	..	..	..
2	21	19	16	14	87.5	14	11	9	81.8	8	6	1	16.7	3	2	0	0
3	18	16	12	9	75	15	12	7	58.3	6	5	2	40	2	1	1	100
4	6	4	4	3	75	3	3	0	0	1	1	0	0	0	..	..	..
Not graded	6	6	4	3	75	6	4	1	25	6	4	1	25	4	2	0	0
Entire series	59	52	42	35	83.3	43	34	21	61.8	22	17	5	29.4	9	5	1	20

* The three year group comprises the patients operated on three or more years prior to the time of inquiry; the five year group, those operated on five or more years prior to the time of inquiry; the ten year group, those operated on ten or more years prior to the time of inquiry, and the fifteen year group, those operated on fifteen or more years prior to the time of inquiry.

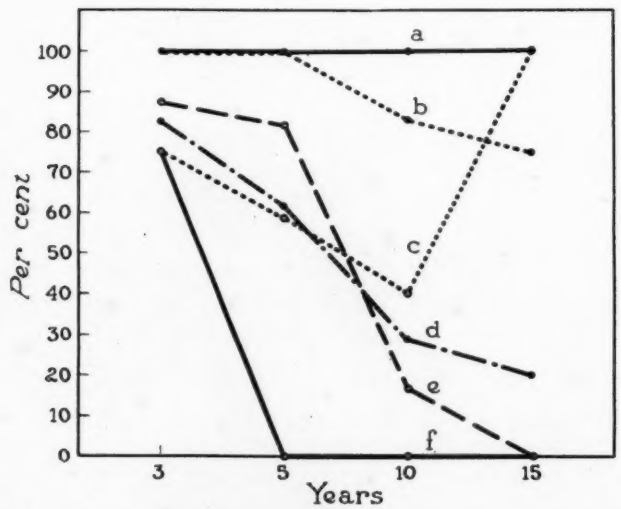

Fig. 2.—Chart showing the percentage of three, five, ten and fifteen year cures of epitheliomas of the eyelids, conjunctiva and cornea of various grades and types in a series of 230 cases: *a*, percentage of cures in cases of squamous cell epithelioma of grade 1; *b*, in cases of basal and squamous cell epithelioma; *c*, in cases of squamous cell epithelioma of grade 3; *d*, in cases of basal cell epithelioma; *e*, in cases of squamous cell epithelioma of grade 2, and *f*, in cases of squamous cell epithelioma of grade 4. Birge.[1,2]

COMMENT

Epitheliomas of the eyelids compose probably from 3 to 5 per cent [8] of the epitheliomas which occur on the body in general. Of these, roughly 25 per cent are of the squamous cell variety. The squamous cell epitheliomas that account for the greatest mortality are those graded 3 and 4, and these make up slightly less than 50 per cent of all the squamous cell epitheliomas of the eye and the eyelid. In order to reduce the mortality from squamous cell epithelioma, attention must be centered on the lesions of grades 3 and 4, which are not often seen. Even with the most vigorous treatment, namely, exenteration, few of the highly malignant lesions (table 3) allow a person to survive long.

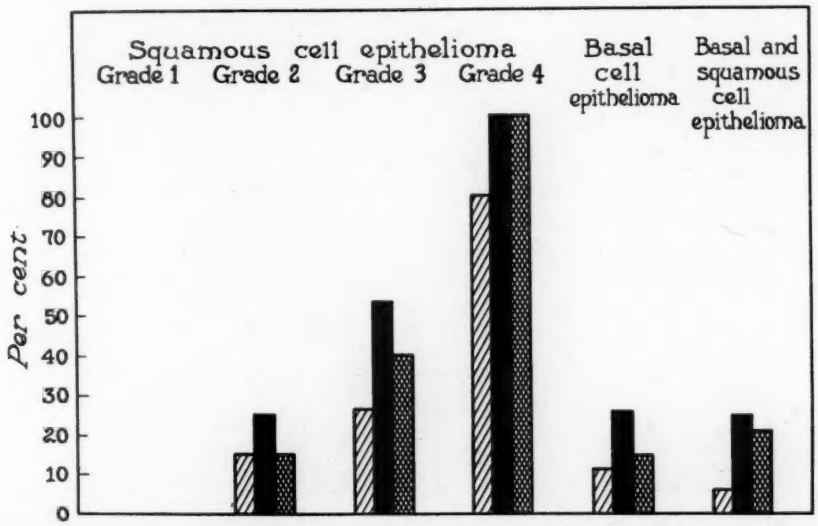

Fig. 3.—Chart showing the relative malignancy of graded squamous cell epitheliomas and other epitheliomas found on the eyelids and external coverings of the eyeball in a series of 230 pathologically classified tumors. The diagonal lines indicate the number of patients who died of cancer of the eye; the solid area, those who became blind as a result of the lesion of the eye, and the crossed lines, those who underwent exenteration.

It would seem that earlier exenteration of growths graded 3 and 4 might offer a means of prolonging the life of persons affected.

Primarily, in all cases in which a tumor of the eye or eyelids is suspected, histologic examination must be made. Secondarily, attention

8. Geschickter, C. F., and Koehler, H. P.: Ectodermal Tumors of the Skin, Am. J. Cancer **23**:804-836 (April) 1935. Broders, A. C.: Squamous-Cell Epithelioma of the Skin, Ann. Surg. **73**:141-160 (Feb.) 1921. Nicolini, R. C.: Cáncer del párpado: consideraciones sobre diagnóstico, pronóstico y tratamiento, Semana méd. **1**:1630-1632 (June 6) 1935; abstracted, Am. J. Cancer **28**:634 (Nov.) 1936.

must be paid to the situation of the tumor on the eye or the eyelids. Lesions in certain places are attended by greater mortality and cause more blindness than lesions in other places. This is evident from glancing at table 4; regardless of histologic grade of malignancy, more deaths and more blindness occurred from lesions of the upper lid. Lesions situated at the inner canthus were nearly as dangerous as those on the upper lid. More exenterations were done on lesions at the inner canthus than elsewhere, perhaps causing a slightly better gross result, in keeping with the more vigorous treatment.

TABLE 4.—*Behavior of Squamous Cell Epitheliomas (Graded) According to the Situation on the Eyelids*

Situation of Growth	Number of Patients		Exenteration		Blind in Affected Eye		Died of Ocular Cancer	
	Total	Traced	No. of Patients	Percentage	No. of Patients	Percentage	No. of Patients	Percentage
Lower lid.....	17	13	4	30.7	4	30.7	2	15.4
Upper lid.....	10	10	4	40.0	6	60.0	4	40.0
Inner canthus.....	8	6	3	50.0	3	50.0	2	33.3
Outer canthus.....	4	3	0	0	0	0	0	0

TABLE 5.—*Behavior of Conjunctival, Corneal and Limbic Squamous Cell Epithelioma on the Basis of Histologic Grading*

Situation of Growth	Number of Patients		Exenteration		Blind in Affected Eye		Died of Ocular Cancer	
	Total	Traced	No. of Patients	Percentage	No. of Patients	Percentage	No. of Patients	Percentage
Conjunctiva.....	11	11	3	27.3	3	27.3	2	18.1
Limbus.....	6	5	1	20.0	2	40.0	0	0
Cornea.....	3	3	0	0	0	0	0	0

Any tumor about the eyelids is likely to prevent perfect closure of the lids and to subject the cornea to exposure and ulceration. A lesion of the upper lip is more likely to cause keratitis from exposure than a lesion of the lower lid. Sometimes this factor has a bearing on the type of treatment chosen; for instance, if plastic repair is contemplated after excision of part of the upper lid, a repair must be done immediately, and this entails the risk of covering some malignant cells, should any remain, with healthy tissue.

Lesions of the outer canthus were rendered exceptionally harmless by excision (table 4). A lesion in this location is more easily completely removed than a lesion at the inner canthus, which, perhaps, tends to invade the orbit more easily.

Lesions situated on the conjunctiva and cornea were not as malignant as were lesions of the upper lid or inner canthus (tables 4 and 5). Corneal lesions, in fact, were as benign as were lesions of the outer canthus.

Primary Corneal Epithelioma.—Primary corneal epithelioma is of sufficient rarity to justify digression here a moment for more full discussion of the 3 cases included in table 5. These growths all involved the cornea primarily. Each of them was associated with a pterygium. Furthermore, in all 3 of the cases some sort of trauma had been inflicted in an attempt to remove the pterygium before the lesion occurred. In 2 of the cases the growth originated at the junction of the pterygium and the normal cornea; in the third, it appeared separated from the advancing edge of the pterygium by approximately 1 mm., so that it may have been connected with some small portion of the pterygium.

Malignant lesions of the cornea, when they occur, are usually of the squamous cell variety, because of the single layer of cuboidal epithelial cells that compose the outer epithelium of the cornea. Sarcoma of the cornea has been reported [9] and probably arises from the corneal stroma, which is mesodermal in origin. Melanosis of the cornea also may occur, secondary to melanotic tumor of the eye; a case has been reported by Schmidt [10] in which the growth occurred eight years after excision of a melanosarcoma of the limbus.

The relation of these 3 cases of corneal squamous cell epithelioma to the trauma inflicted in an attempted removal of a pterygium is in keeping with the generally accepted idea that epitheliomas arise in many cases from chronic irritation.

Corneal squamous cell epitheliomas are generally relatively benign, in spite of the high grade of malignancy seen under the microscope (table 1). In 1 of these cases the growth was graded 2, and in the other 2 the growths were graded 3, yet in none of them was blindness or death the result (table 5).

Limbic Epithelioma.—Squamous cell epitheliomas that arise at the corneal limbus appear to be less frequent than those on the bulbar or palpebral conjunctiva (table 1, fig. 1), but they are more frequent than primary corneal epitheliomas. The growths were all graded either 1 or 2, and while none of the 5 patients who were traced died, an eye was lost in 40 per cent (table 5). Typical limbic epitheliomas are shown in figure 4 *a* and *b*. The growth in figure 4 *a* probably represents a secondary involvement of the limbus, while that in figure 4 *b* represents a growth which may have been a primary limbic epithelioma.

9. Gastew, A. A., and Werpoukhowsky, M. M.: Sarcome de la cornée, Ann. d'ocul. **172**:587-595 (July) 1935.

10. Schmidt, R.: Isolierte Melanosis corneae, 8 Jahre nach Exzision eines oberflächlichen Melanosarkoms am Hornhautlimbus, Klin. Monatsbl. f. Augenh. **93**: 164-170, 1934.

Epitheliomas in this region usually are fed by rather large vessels [11] and tend to grow rapidly. Often they are thought to be caused by injury. The discomfort of the sensation of the foreign body makes the patient seek early consultation, and consequently malignant lesions in this region are likely to be seen early.

Conjunctival Epithelioma.—The conjunctiva of the globe or lids is frequently involved by squamous cell epithelioma. In this series less blindness was caused by lesions of the conjunctiva than by lesions at the limbus. Conjunctival lesions, however, caused a higher percentage of deaths than did lesions of the lower lid, the outer canthus or the cornea.

In this series none of the conjunctival lesions was of the basal cell type. Mazzi,[12] however, has reported a rare case of basal cell epithelioma on the conjunctiva of each eye. Lopes de Andrade [13] reported bilateral squamous cell epithelioma of the conjunctiva, but in none of the cases in this series did the growth occur bilaterally.

Conjunctival squamous cell epitheliomas are shown in figure 4 *c* and *d*. Lesions of the conjunctiva may be highly malignant, and it is important to have a histologic estimation of the malignancy before making a final decision as to the method of treatment.

Recurrence and Metastasis.—Recurrences after treatment are common with all types of carcinoma of the eye and eyelids. Several factors probably contribute to this fact. Among them is the desire to save the eye and at the same time to cure the lesion. This tends to influence the clinician to choose the minimal treatment rather than a safer method of removal of cancer, which may entail removal of the eye. Another factor influencing the frequency of recurrence is the thought that many of the lesions are essentially benign and consequently do not need histologic examination. Many of the patients in this series came in with recurrences after treatment by unacceptable methods, such as the use of salves, escharotics and out and out quack cancer "cures."

In this group of cases more than 50 per cent of the patients had recurrences after one or another form of treatment. This percentage is based on all patients who received any sort of treatment after the cancer appeared. Many of them were not seen at the clinic until comparatively late. Lesions of grade 3 had the greatest tendency to recur (table 2).

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11. Benedict, W. L.: Epithelioma of the Limbus, S. Clin. North America **9**: 813-822 (Aug.) 1929.

12. Mazzi, L.: Epitelioma epibulbare bilaterale simmetrico, Arch. di ottal. **41**:260-273 (June-Nov.) 1934; abstracted, Am. J. Cancer **28**:815 (Dec.) 1936.

13. Lopes de Andrade, A.: Épithéliome bilatéral du limbe scléro-cornéen, Ann. d'ocul. **172**:897-907 (Nov.) 1935.

The greatest danger from lesions about the eyes is that of extension to the orbit. The majority of squamous cell lesions about the eyes drain into the orbit before the lymph nodes of the face or the neck are affected. Lesions of the cornea or conjunctiva almost always extend into the orbit rather than down to the facial nodes.

Metastasis to the lymph nodes of lesions of the lower lids usually extends first to the submaxillary glands, and the metastatic growths

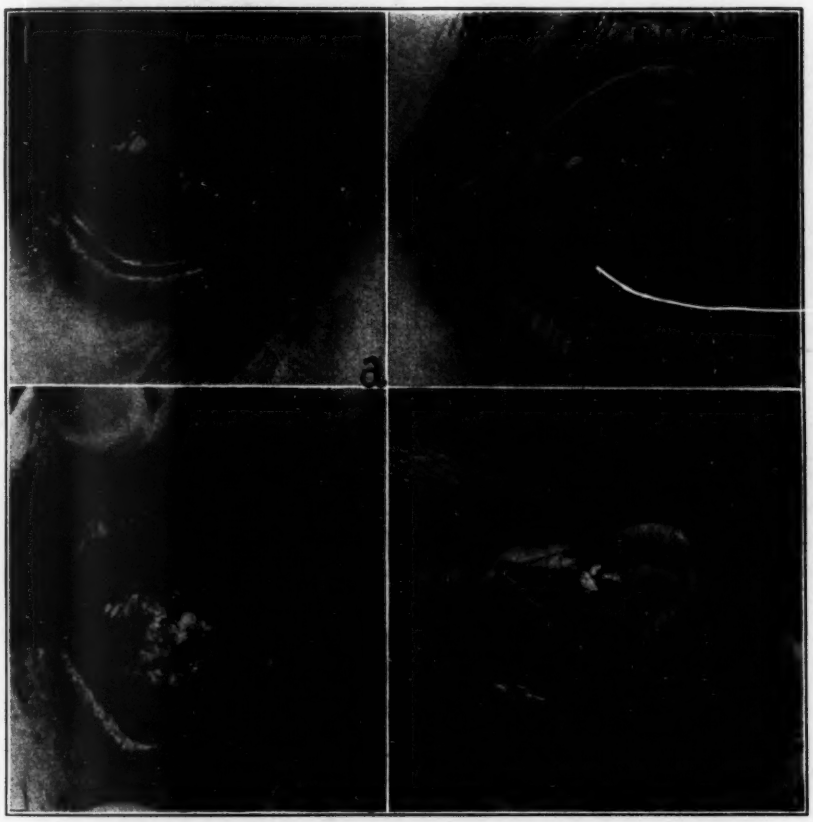

Fig. 4.—Squamous cell epitheliomas: *a, b* and *c,* squamous cell epithelioma of the conjunctiva and the corneal limbus, and *d,* a squamous cell epithelioma of the conjunctival surface of the tarsus and the upper lid.

are found under the angle of the jaw. The cervical nodes will not be affected early. Lesions of the upper lid usually drain into the preauricular nodes or into small nodes in the temporal region. In this series treatment of such metastatic growths consisted of irradiation.

The greater the malignancy, the greater is the chance of involvement of either the lymph nodes or the orbit, regardless of treatment (table 2).

Of lesions of the squamous cell type about the eye, 30 per cent of those of grade 2, 60 per cent of those of grade 3 and 100 per cent of those of grade 4 invaded the orbit.

Blindness from squamous cell lesions of the eyelids, conjunctiva and cornea is directly proportional to the grade of malignancy (table 2). There is more blindness from squamous cell lesions as a whole and from individual grades of such lesions, except those of grade 1, than there is from any other type of epithelioma of the eye or eyelids.

The chance of saving the eye in the presence of lesions of the eye and adnexa is about 50 per cent if the lesions are of grade 3 and zero if they are of grade 4. With basal cell lesions of this type, not considering the situation of the lesion on the lids, which is of considerable importance, there is generally about a 75 per cent chance of saving the eye.

The mortality rate of epidermoid carcinoma of the eyelids and conjunctiva in general is about 12.2 per cent,[1] but this does not hold true of the individual grades of squamous cell epithelioma. The correlation between the various grades of malignancy and the span of individual life is shown in figure 2.

While squamous cell epithelioma of grade 4 caused the death of 80 per cent of the patients in this small series and that of grade 3 caused the death of 46.7 per cent, there must have been many cases of epithelioma of a low grade of malignancy to give a general mortality of 12.2 per cent in 464 cases. In the majority of cases growths of a low grade of malignancy do not prove fatal unless treatment is neglected, but the growths that persistently cause death are those of a high grade of malignancy (fig. 3). These are fatal regardless of treatment, and apparently the best way of reducing the mortality from them is by earlier and more extensive treatment.

Most carcinomas develop over a long period,[14] during which there is plenty of opportunity for treatment. Simmons [15] expressed the belief that the frequent recurrence of cutaneous cancer is due to failure to excise enough tissue around the lesion and failure otherwise to follow recognized principles of surgical technic. Belgeri [16] stated that inadequate local treatment causes loss of valuable time and plays havoc with the prognosis. Although these statements are true in some instances, there is another more important fact. Most of the malignant growths

14. (*a*) Figi, F. A.: Epithelioma of the Lower Lip: Results of Treatment, Surg., Gynec. & Obst. **59**:810-819 (Nov.) 1934. (*b*) Birge.[1] (*c*) Benedict.[11]

15. Simmons, H. T.: Surgery in the Treatment of Primary Skin Carcinoma, Lancet **2**:938-940 (Oct. 26) 1935.

16. Belgeri, F.: Tumores malignos del aparato visual su diagnóstico y su tratamiento, Semana méd. **1**:1048-1055 (April 11) 1935; abstracted, Am. J. Cancer **28**:814 (Dec.) 1936.

around the eyelids that cause either blindness or death are those of a high grade of malignancy or those of which treatment has been greatly neglected.

Carcinoma about the eyelids can be cured in most cases if removal is effected early enough. The eyelids are constantly in view. Yet the average duration of most of the lesions when the patients seek treatment is well over a year; Figi [14a] reported that the average duration of lesions of the lips before the patient comes for treatment is twenty-eight and three tenths months. MacCarty [17] has shown that the average size of surgically excised malignant growths has not diminished in the past fourteen years. It is entirely possible that removal of all tumors of the eyelids, benign or malignant, when first seen, would reduce the amount of blindness and the mortality from carcinoma of the eyelids.

REPORT OF CASES

Photomicrographs will not be reproduced here because the grading of squamous cell epitheliomas has been amply covered in the literature and the lesions on which this paper is based had the same cellular structure as have other squamous cell epitheliomas.

CASE 1.—A man aged 57 had a growth on the right eye near the limbus, measuring 6 by 3 by 2 mm., which involved the cornea (fig. 4 *a*). It was excised and cauterized. The histologic diagnosis was squamous cell epithelioma of grade 1. Vision in the right eye before excision was 6/6 and in the left eye 6/5. Shortly afterward, reexamination showed the vision to be the same.

CASE 2.—A man aged 59 came to the clinic because of a growth on the conjunctiva of the left eye, near the limbus (fig. 4 *b*). This had been noted six weeks before admission, and the condition had grown steadily worse. Vision in the right eye was 6/20 and in the left eye 6/12. The mass was excised, and a histologic diagnosis of squamous cell epithelioma of grade 2 was made. Over the course of a year the epithelioma was treated with 3,050 milligram hours of radium, the eye being screened by 2 mm. of lead. At the end of this time vision in the right eye was 6/12, and in the left eye 6/10.

CASE 3.—A man aged 50 came to the clinic because of a small clear tumor on the inner side of the right cornea, which he had noticed ten years before his admission. Six weeks before admission it had begun to grow (fig. 4 *c*). A histologic diagnosis of squamous cell epithelioma of grade 2 was made. The epithelioma was excised from the conjunctiva and cornea; fulguration was done, and the wound was left open. One year later vision in the right eye was 6/10 and in the left eye 6/7.

CASE 4.—A mass suddenly developed in the right upper eyelid of a man aged 25 (fig. 4 *d*). The mass had appeared one year before he came to the clinic. It had been opened as a cyst three times but had become progressively worse. Radium was applied. At the clinic, vision in the right eye was 6/6 and in the

17. MacCarty, W. C.: The Size of Operable Cancers (a Study of 7,179 Specimens), Am. J. Cancer **17**:25-33 (Jan.) 1933.

left eye 6/7. Histologic examination disclosed inflammatory tissue. The mass recurred six months after the radium therapy and was removed; this time the histologic diagnosis was squamous cell epithelioma of grade 2. The growth was treated with 1,400 milligram hours of radium, a 1.5 mm. lead filter being used. Two months later a small tumor grew from the temporal side of the right upper eyelid. It was excised, and 25 milligram hours of radium was given. The histologic diagnosis was inflammatory tissue.

Three months after the foregoing excision, the tumor recurred and measured 12 by 18 mm. Excision was done, and plasma cell infiltration of inflammatory tissue, with amyloid degeneration, was found. Radium was again applied, 1,400 milligram hours, and the eye was screened with a shield composed of 2 mm. of lead and 1.5 mm. of monel metal. A 25 mg. plaque, kept in motion thirty minutes, was applied.

Four months later another recurrence was noted, and 1,400 milligram hours of radium was applied, a shield of 2 mm. of lead and 1.5 mm. of monel metal being placed 2.5 cm. from the right orbit. A plaque of 25 mg. was kept in motion for thirty minutes.

Six months elapsed, and then another ulcer, or a recurrence, developed, which was cauterized. Histologic examination revealed that glands deep in the lid had a tendency to form squamous cell epithelioma.

Four months subsequent to this examination, or three years after the growth was first noticed, vision in the right eye was 6/6 and in the left eye 6/5. There was no sign of a new growth, and there was only moderate contraction of the right upper lid, with no adhesions or symblepharon. The patient is still being followed.

CASE 5.—A woman aged 53 came to the clinic because of a growth on the right upper eyelid (fig. 5 *a*). She had first noticed the growth five years before; it had varied in size from time to time. For five months she had noticed a stinging sensation. A pathologic diagnosis of squamous cell epithelioma of grade 2 was made. The lesion was excised, and the skin flaps were drawn together.

CASE 6.—A woman 51 years of age came to the clinic because she had had a tumor on the right upper eyelid for one year (fig. 5 *b*). It had broken down occasionally and had never healed. There was no pain. Vision in the right eye was 6/6 and in the left eye 6/6. The mass measured 1 by 1 by 0.5 cm. and was ulcerated on the conjunctival surface. It hung down over the pupillary area and obstructed vision. It was excised under local anesthesia and was found to be squamous cell epithelioma of grade 3. Treatment consisted of 1,112 milligram hours of radium and the placement of three needles in the skin, equaling 1,040 milligram hours of radium.

Six years later the growth recurred and was excised so widely that the lids had to be sutured in an attempt to save the eye. This time the growth was a mixed squamous cell epithelioma and an adenocarcinoma of grade 4. Radium, 2,400 milligram hours, was applied, a shield composed of 2 mm. of lead and 1.5 mm. of monel metal being placed 2.5 cm. from the right upper lid.

CASE 7.—A man 44 years of age had had a growth on the left upper lid for three years (fig. 5 *c*). It had gradually increased in size. He had been given treatment with radium after two years. When he was seen at the clinic, vision in his right eye was 6/15 and in his left eye 3/60. There was osseous involvement, and the preauricular and submaxillary glands were enlarged. Exenteration was done, and the malignancy of the squamous cell epithelioma was graded 3. Radium

was applied, 2,400 milligram hours, the eye being screened with a shield composed of 2 mm. of lead and 1.5 mm. of monel metal.

CASE 8.—A man aged 56 nine months before seeking treatment noticed a growth on the side of his nose (fig. 5 *d*). Pastes had been applied for six months. Radium had been used for three months. Vision in the right eye was 6/20 and in the left eye 6/30. The submaxillary glands were enlarged. The man had diabetes. The histologic diagnosis was squamous cell epithelioma of grade 3. Exenteration was done, and the patient lived for five months.

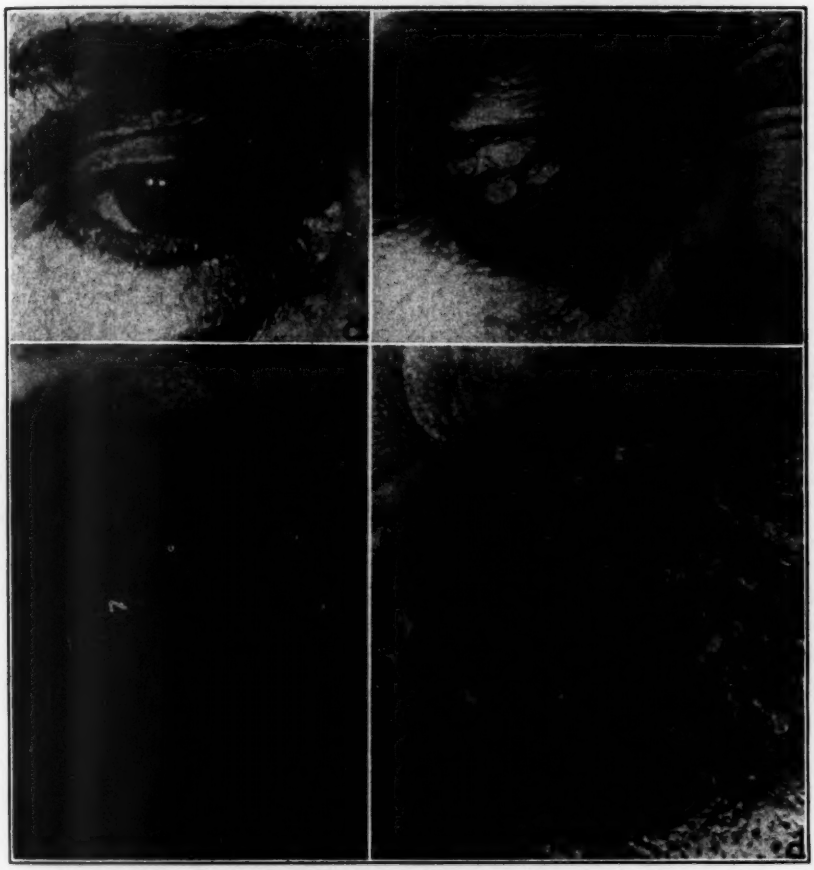

Fig. 5.—Squamous cell epitheliomas: *a* and *b*, comparatively early squamous cell epithelioma of the inner canthus and of the upper lid; *c*, a late lesion of the outer canthus, and *d*, an early but invasive squamous cell lesion of a diabetic patient.

SUMMARY AND CONCLUSIONS

Squamous cell epithelioma of the eyelids, conjunctiva and cornea occurred in 25 per cent of the cases of epithelioma of the eye and its adnexa that were studied. Thirty-seven per cent of the squamous cell epitheliomas arose from the conjunctival surfaces, including the cornea. Primary corneal squamous cell epithelioma occurred in 5 per cent of the

cases in this series. The malignancy of most of the lesions in the entire series was either of grade 2 or grade 3 on the basis of histologic examination. Chronic irritation or trauma was an etiologic factor in 32.2 per cent of the cases.

There was close correlation between the clinical behavior over a period of fifteen years and the grade of malignancy. Lesions of grade 1 did not cause death or loss of an affected eye. Lesions of grade 2 caused loss of the affected eye in 25 per cent of cases and death in 15 per cent. Lesions of grade 3 caused loss of the affected eye in 53 per cent of cases and death in 46 per cent. Lesions of grade 4 caused loss of the affected eye in all cases and death in 80 per cent.

The situation of the lesion about the eye or eyelids is of considerable importance, judged by the related percentage of mortality and blindness. Recurrences were frequent in this series. The average mortality of all types of epithelioma of the eye and eyelids was about 12 per cent. Mortality and blindness were directly proportional to the histologic grade of malignancy. Lesions of grades 3 and 4 were responsible for the largest part of the mortality and blindness.

Given (1) early recognition of the grade of malignancy and (2) treatment proportional with the grade of malignancy, carcinomas about the eye should carry low mortality.

TREATMENT OF STAPHYLOCOCCIC CONJUNCTIVITIS WITH STAPHYLOCOCCUS TOXOID

PRELIMINARY NOTE

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In a previous communication [1] toxin-producing staphylococci were brought forward as an important cause of chronic catarrhal conjunctivitis. Characteristic of conjunctivitis believed to be staphylococcic is the failure of locally applied antiseptics to heal it or to rid the eye of the offending bacteria. This failure is believed to be related to the predilection of staphylococci for the skin and glands of the margins of the lids; here the bacteria are protected from the action of antiseptics and are able to reinfect the conjunctiva.

In view of the efficacy of immunization with staphylococcus toxin in protecting rabbits against instillations of toxin which would produce severe conjunctivitis in normal rabbits,[1] an attempt has been made to obtain healing in human beings with conjunctivitis by immunization. Although good results with crude toxin have been reported by Burky,[2] toxoid was employed in the present study because of its greater safety and availability. The Lederle Laboratories made available a supply which, according to the report of Blair and Hallman,[3] could be expected to produce a notable rise in the antitoxin titer after a single course.

The present report records the results obtained in the treatment of 57 patients with conjunctivitis harboring staphylococci the toxin-producing ability of which was either tested on the rabbit conjunctiva or presumed from associated presumptive tests. No other possible etiologic factor could be found in 46 of the patients; of the remaining 11, 3 had a conjunctival eosinophilia suggesting an associated allergy, 5 had mild acne rosacea and 3 had cicatricial trachoma of questionable activity. In order to minimize as far as possible the factor of spontaneous healing, only persons were chosen whose conjunctivitis had resisted local

From the Department of Ophthalmology, College of Physicians and Surgeons, Columbia University, and the Institute of Ophthalmology, Presbyterian Hospital.

1. Thygeson, P.: Bacterial Factors in Chronic Catarrhal Conjunctivitis: Role of Toxin-Forming Staphylococci, Arch. Ophth. **18**:373 (Sept.) 1937. Allen, J.: Am. J. Ophth. **20**:1025, 1937.

2. Burky, E. L.: Am. J. Ophth. **19**:841, 1936.

3. Blair, J. E., and Hallman, F. A.: Proc. Soc. Exper. Biol. & Med. **34**: 637, 1936.

No.	Patient	Type of Conjunctivitis	Type of Hemolytic Staphylococcus	Results of Toxoid Therapy: Clinical	Results of Toxoid Therapy: Bacteriologic (Cultures at Last Examination)
37	W. U.	Chronic conjunctivitis (acne rosacea)	Aureus	Healing	—
33	R. H.	Chronic keratoconjunctivitis	Aureus	Healing	—
78	J. P.	Chronic keratoconjunctivitis	Aureus	Healing	—
1a	A. C.	Chronic blepharoconjunctivitis with ulcers	Aureus	Healing	—
2a	A. M.	Chronic blepharoconjunctivitis with ulcers (10 yr.)	Aureus	Healing	—
3a	J. L.	Chronic blepharoconjunctivitis	Aureus	Healing	—
1	H. E. S.	Chronic keratoconjunctivitis	Albus	Healing	+
28	G. A.	Trachoma III-IV	Aureus	Healing	+
372	J. S.	Chronic catarrhal conjunctivitis	Aureus	Healing	+
420	S. R.	Chronic blepharoconjunctivitis	Aureus	Healing	+
506	R. K.	Chronic blepharoconjunctivitis	Aureus	Healing	+
9	E. H. J.	Chronic conjunctivitis with meibomitis	Albus	Healing	No record
43	E. G.	Chronic conjunctivitis with recurrent styes	Aureus	Healing	No record
25	P. T. W.	Chronic blepharoconjunctivitis	Aureus	Healing; recurrence; healing	+ —
216	M. R.	Chronic blepharoconjunctivitis	Aureus	Healing; recurrence; healing	— +
6	B. K.	Chronic blepharoconjunctivitis	Aureus	Healing; recurrence; healing	+ +
18	J. P.	Chronic blepharoconjunctivitis	Aureus	Healing; recurrence; healing	+ +
40	F. W.	Chronic keratoconjunctivitis	Aureus	Healing; recurrence; healing	+ +
55	H. R.	Chronic keratoconjunctivitis with styes	Aureus	Healing; recurrence; healing	+ +
538	H. M.	Chronic keratoconjunctivitis	Aureus	Healing; recurrence; healing	+ +
622	R. E.	Chronic keratoconjunctivitis	Aureus	Healing; recurrence; healing	+ +
37	E. R.	Chronic catarrhal conjunctivitis	Aureus	Improvement	—
39	M. H.	Chronic catarrhal conjunctivitis	Albus	Improvement	—
56	J. H.	Chronic blepharoconjunctivitis	Aureus	Improvement	—
59	G. B.	Chronic catarrhal conjunctivitis	Albus	Improvement	—
61	R. B.	Chronic catarrhal conjunctivitis	Albus	Improvement	—
70	J. B.	Chronic catarrhal conjunctivitis	Aureus	Improvement	—
94	J. K.	Chronic catarrhal conjunctivitis	Albus	Improvement	—
105	D. R.	Chronic blepharoconjunctivitis	Aureus	Improvement	—
107	L. A. W.	Chronic blepharoconjunctivitis	Albus	Improvement	—
244	C. D.	Chronic blepharoconjunctivitis	Aureus	Improvement	—
250	R. G.	Chronic blepharoconjunctivitis	Aureus	Improvement	—
286	J. D.	Chronic keratoconjunctivitis (acne rosacea and seborrheic dermatitis)	Aureus	Improvement	—
4	S. N.	Chronic catarrhal conjunctivitis	Aureus	Improvement	+
23	G. M. A.	Chronic catarrhal conjunctivitis (eosinophilia)	Aureus	Improvement	+
54	H. K.	Chronic catarrhal conjunctivitis	Aureus	Improvement	+
83	W. L.	Chronic keratoconjunctivitis	Albus	Improvement	+
103	J. B.	Trachoma III and IV	Aureus	Improvement	+
239	N. M.	Chronic blepharoconjunctivitis (sycosis vulgaris)	Aureus	Improvement	+
405	E. T.	Chronic catarrhal conjunctivitis with recurrent styes	Aureus	Improvement	+
48	M. J.	Chronic catarrhal conjunctivitis (chalazia)	Albus	No improvement	—
52	H. C.	Chronic catarrhal conjunctivitis	Albus	No improvement	—
72	I. K.	Chronic keratoconjunctivitis	Aureus	No improvement	—
87	B. S.	Chronic catarrhal conjunctivitis	Aureus	No improvement	—
135	F. M.	Chronic keratoconjunctivitis	Albus	No improvement	—
11	I. G.	Chronic catarrhal conjunctivitis (acne rosacea)	Albus	No improvement	+
19	M. S.	Chronic blepharoconjunctivitis (sycosis vulgaris)	Aureus	No improvement	+
21	R. S.	Chronic conjunctivitis (acne rosacea)	Aureus	No improvement	+
112	H. W.	Chronic catarrhal conjunctivitis	Aureus	No improvement	+
119	D. H.	Chronic catarrhal conjunctivitis (sensitivity to drug)	Aureus	No improvement	+
149	E. W.	Chronic blepharoconjunctivitis (eosinophilia)	Aureus	No improvement	+
271	V. S.	Chronic keratoconjunctivitis	Aureus	No improvement	+
404	M. W.	Chronic blepharoconjunctivitis	Albus	No improvement	+
486	J. P.	Trachoma III and IV	Aureus	No improvement	+
494	A. C. H.	Chronic blepharoconjunctivitis	Aureus	No improvement	+
557	M. G.	Chronic blepharoconjunctivitis	Albus	No improvement	+
152	D. G.	Chronic catarrhal conjunctivitis	Albus	No improvement	No record

treatment not less than two months. The majority of the patients had had conjunctivitis for over a year.

The toxoid was given biweekly, starting with an initial injection of 0.01 cc. of dilution no. 2. The dose was increased to a maximum of 1 cc. in steps of 0.02, 0.04, 0.06, 0.08, 0.1, 0.2, 0.4, 0.6 and 0.8 cc. In the few cases in which general reactions occurred, this schedule was modified. Injections of less than 0.5 cc. were given intradermally, and the remainder were given subcutaneously. In those cases in which noticeable improvement did not occur on completion of the series, injections of 0.5 cc. were continued at weekly intervals for at least six weeks. In all cases the same local treatment which had previously proved ineffective was continued.

The results of treatment are recorded in the table. Healing occurred in 21 of the 57 patients, 19 showed clinical improvement, and the condition of 17 remained unchanged. The cultures of 24 of the 57 patients became negative. There was a recurrence of symptoms after periods varying from four to six months in 8 of the 21 persons in whom clinical healing occurred. In all of these the symptoms disappeared after a second course of toxoid therapy. This recurrence was probably related to the fact that in 7 of the 8 patients some toxigenic staphylococci were still present after healing. The cultures of the 13 patients in whom healing occurred, and who have as yet had no recurrence, were negative after healing.

Before the favorable results are credited to the production of a specific resistance to staphylococcus exotoxin through the formation of antitoxin, the following factors must be considered: (1) nonspecific protein effect, (2) spontaneous healing and (3) effect of local treatment. There seems to be no way of evaluating the first factor, but the second and third would appear to be of minor importance, since the series included only persons with conjunctivitis of long standing which had long resisted local treatment.

SUMMARY AND CONCLUSIONS

A series of 57 patients with conjunctivitis presumably due to toxigenic staphylococci were treated by immunization with staphylococcus toxoid. The conjunctivitis was of long standing and had resisted local treatment for periods of not less than two months. Twenty-one of the 57 patients showed healing, 19 showed clinical improvement and 17 showed no improvement. The cultures of only 24 of the 57 patients became negative after treatment. Eight of the 21 patients in whom healing occurred had a recurrence of symptoms; these disappeared after a second course of toxoid therapy. The recurrences appeared to be related to the failure of treatment to eliminate the offending bacteria. It is believed that the results obtained warrant further clinical trial of staphylococcus toxoid.

MANNITOL FERMENTATION AS AN INDICATOR OF CONJUNCTIVAL PATHOGENICITY OF STAPHYLOCOCCI

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The recent reports of Burky,[1] Thygeson,[2] Allen [3] and O'Brien and Allen [4] indicate an increasing recognition of the important role of toxin-producing staphylococci in conjunctival and corneal inflammations. The common occurrence of saprophytic varieties of staphylococci on the margins of the lids and on the conjunctiva necessitates some test by which toxigenic strains may be recognized readily. Any strain can be tested for its ability to elaborate a conjunctivitis-producing toxin by a method which has been previously described,[2] but this is a time-consuming and expensive procedure and not adapted to routine use.

Of the simple tests which have been shown to have some correlation with the production of toxin, the mannitol fermentation test appears to permit of widest application. Overnight readings can be obtained on phenol red–mannitol agar plates inoculated directly from the conjunctiva, a distinct advantage over the coagulase and crystal violet agar tests which require secondary inoculations. In a former study [2] a correlation between the ability of staphylococci to ferment mannitol and their ability to produce a conjunctival toxin was found in only 52 of 57 strains, or 91.2 per cent, and of 57 strains producing no conjunctival toxin, 9, or 15.7 per cent, fermented mannitol. These tests, however, were made by the tube method, which subsequent work has shown to be less reliable than the plate method.

In the present study 196 strains of staphylococci from eyes with conjunctivitis were tested for ability to ferment mannitol and for ability to form a conjunctivitis-producing toxin. The toxins were prepared according to the method of Leonard and Holm [5] and tested on the

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1. Burky, E. L.: Am. J. Ophth. **19**:841, 1936.
2. Thygeson, P.: Bacterial Factors in Chronic Catarrhal Conjunctivitis: Role of the Toxin-Forming Staphylococci, Arch. Ophth. **18**:373 (Sept.) 1937.
3. Allen, J. H.: Am. J. Ophth. **20**:1025, 1937.
4. O'Brien, C. S., and Allen, J. H.: Am. J. Ophth. **21**:641, 1938.
5. Leonard, G. F., and Holm, A.: J. Immunol. **29**:209, 1935.

conjunctiva of rabbits, as described in a previous report.[2] The mannitol fermentation test was carried out by streaking the culture on phenol red–mannitol agar (Difco) plates,[6] and the reaction was considered positive if after incubation overnight the medium around the colonies had changed from pink to yellow. Of the 92 strains which showed definite production of toxin, all but 1 fermented mannitol, and of the 104 nontoxigenic strains, only 4 showed fermentation. These results (a correlation of 97.4 per cent) compared favorably with those obtained with the coagulase test (considered by most workers to be the most reliable indicator of pathogenicity) for this series, all but 1 of 86 strains tested giving positive reactions and all but 2 of 82 nontoxigenic strains giving negative reactions (a correlation of 98.2 per cent).

The mannitol fermentation test, as performed on phenol red–mannitol agar plates, would seem therefore to be a reliable indicator of probable conjunctival pathogenicity of staphylococci. It is employed routinely in the studies on conjunctivitis now being conducted at the Vanderbilt Clinic.

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6. This medium was called to my attention by Mr. George H. Chapman, New York.

RECURRENT RETINAL AND VITREOUS HEMORRHAGES IN THE YOUNG— EALES' DISEASE

REPORT OF TWO CASES

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The first adequate description of recurring retinal hemorrhages in young adults was given by Henry Eales in 1880.[1] In 1882 [2] he further summarized the characteristics of his earlier reported cases in a paper entitled "Primary Retinal Haemorrhage in Young Men." His first impression was that all patients with this condition suffer from severe constipation and epistaxis. He later elaborated on this theory in the following manner:

> From the absence of any evidence of any of the various blood conditions known to cause haemorrhage; from the absence of albuminuria, diabetes, gout, syphilis, and of any high arterial tension; from the character of the haemorrhage, and from the evidence of local variations of circulation; and from the slow pulse, constipation, flushing of the face, headache, and puffiness and discolouration of the eyes, I am inclined to attribute this combination of conditions to a neurosis affecting both the circulatory organs and the digestive system, leading on the one hand to partial inhibition of the muscular movements of the bowels, and to a vaso-motor contraction of the vessels of the alimentary canal, with inhibition of its secretory functions, thereby causing dyspepsia, constipation, malnutrition; and on the other hand to a compensatory dilatation of the systemic capillaries, especially those of the head, and, in these cases, of the retina, causing over-distention of the venous system and systemic capillaries, with liability to rupture on the occurrence of any intensifying cause. Hence the headache, the epistaxis, the retinal haemorrhages, the tortuosity and fullness of the retinal vessels, and temporal artery.

It is interesting to note that in Eales' series of cases the left eye was most frequently affected. He attributed this to the anatomic condition of the vessels; that is, the vessels going to the right side take a more circuitous course and do not have a direct extension from the aortic arch. Both factors favor a higher vascular tension on that side.

From the ophthalmologic service of the Manhattan Eye, Ear and Throat Hospital.

Read at the Seventh Congress of the Pan American Medical Association, Jan. 20, 1938.

1. Eales, H.: Cases of Retinal Haemorrhage with Epistaxis and Constipation, Birmingham M. Rev. **60**:262-273, 1880.

2. Eales, H.: Primary Retinal Haemorrhage in Young Men, Ophth. Rev. **1**:41, 1882.

INCIDENCE

This interesting observation concerning the incidence of the condition in the left eye is borne out by the numerous case reports appearing in the literature [3] and by 5 cases which I observed personally, in which the hemorrhages usually began in the left eye. The right eye when involved does not as a rule show the same severity of the disease. That this condition is rare may be deducted from a study of the literature and from my personal observation in the clinics of the Wilmer Ophthalmological Institute and at the Manhattan Eye, Ear and Throat Hospital. At the Manhattan Eye, Ear and Throat Hospital, where about 29,000 persons with ophthalmic conditions are treated a year, only 6 cases of typical Eales' disease have been brought to my attention. In all I have observed 12 cases of this rare condition. Eales stated that he saw only 1 patient with the condition among 12,000 patients treated for ocular diseases.

It is generally agreed that the condition has a predilection for the male sex,[4] although many typical cases occurring in young females have been reported.

ONSET

The hemorrhages occur spontaneously and usually have an acute onset, not associated with exertion. Some patients complain of a slight blurring of their vision for a few days before the attack, which they usually attribute to autointoxication or eyestrain. The hemorrhages are not related to hemorrhages elsewhere in the body, though epistaxis has been reported. All the patients are in apparent good health, though some complain of a slightly depressed feeling, with sluggishness of the bowels or severe constipation. About the only ophthalmoscopic sign concerning which there seems to be a general agreement is that there is a noticeable distention of the veins at the time the hemorrhages occur.[5]

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3. Davis, A. E.: Recurrent Retinal Hemorrhage of Adolescence, Am. J. Ophth. **3**:657, 1920. Kennedy, W. A., and Wagener, H. P.: Recurrent Idiopathic Retinal Hemorrhages, Am. J. Ophth. **17**:532, 1934. Kokott, W.: Klinische Untersuchungen über die Aetiologie der juvenilen rizidivierenden Glaskörperhämorrhagien, Klin. Monatsbl. f. Augenh. **94**:327, 1935. Löwenstein, A.: Zur Klinik der Augentuberkulose: Zur Frage des Ursprungs der rezidivierenden Glaskörper- und Netzhautblutungen jugender Individuen, ibid. **95**:458, 1935.

4. Moore, R. F.: Medical Ophthalmology, London, J. & A. Churchill, 1925, p. 243. von Hippel, E.: Zur Frage der Perivasculitis retinae (recidivierende Glaskörperblutungen bei Jugendlichen), Arch. f. Ophth. **134**:121, 1935. Adams P. H.: Discussion on the Significance of Retinal Haemorrhages, Tr. Ophth. Soc. U. Kingdom **42**:216, 1922.

5. Lyle, T. R.: Vascular Diseases of the Retina, Proc. Roy. Soc. Med. **29**: 391, 1936. Pavia, J. L., and Dusseldorp, M.: Flebitis y hemorrhagias de retina en tuberculosos, Rev. oto-neuro-oftal. **8**:7, 1933.

In one of my own cases this distention was noticeable for two years, and only after the last attack subsided did the veins begin to have a normal appearance. During this two year interval there had occurred at least eight attacks of hemorrhage in the vitreous, the vision being reduced to perception of hand movements at times. The distention of the veins always appeared greatest just previous to an attack, though of course the fundus could not be studied at all times, owing to the severity of the hemorrhages.

TYPES OF HEMORRHAGES

It may be roughly stated that hemorrhages in the retina and vitreous are the result of two chief causes: an altered condition of the blood itself or a diseased condition of the walls of the blood vessels. The hemorrhages seen in Eales' disease are usually confined to the periphery of the retina, being found only occasionally near the disk, and then only around the larger venous trunks. In some cases retinal exudates have been present in addition. Flame-shaped hemorrhages are rarely seen, although they may be present. The fovea and macula are nearly always spared. The hemorrhages usually extend anteriorly into the vitreous, and in 1 case that I am reporting they were entirely confined to the vitreous. Though hemorrhages in the vitreous are usually profuse, often blotting out the entire vision, it is surprising how completely they will absorb, with a return of normal vision.

CAUSES OF HEMORRHAGES

Hemorrhages in the retina or vitreous are always of traumatic, toxic or infective origin. Probably the most difficult factor to be ruled out is a focal infection, such as an infection of the teeth, tonsils or sinuses. Syphilis, diabetes and cardiovascular renal disease must also be completely ruled out.[6] Hypothyroidism and hyperthyroidism, often associated with a calcium deficiency brought about by a diminished parathyroid secretion, have been fully discussed by Zentmayer,[7] Young[8] and Jeandelize.[9] That this condition often occurs at the time of puberty

6. Ellett, E. C.: Sequels of Vitreous Hemorrhages, Am. J. Ophth. **6**:496, 1923. Redding, L. C.: Recurrent Hemorrhages into the Retina and Vitreous in Young Persons, Atlantic M. J. **27**:640, 1924. Radcliffe, McC., and Young, C. A.: Recurrent Hemorrhages in the Vitreous, Am. J. Ophth. **6**:496, 1923.

7. Zentmayer, W.: Recurrent Retinal Hemorrhages, Am. J. Ophth. **3**:652, 1920.

8. Young, C. A.: Recurrent Hemorrhages into the Retina and Vitreous: Calcium Deficiency as Possible Cause, Tr. Am. Acad. Ophth. **34**:191, 1929.

9. Jeandelize, P.; Bretague, P., and Richard, G.: Hémorragies récidivantes du vitré et hyperthyroïdie, Ann. d'ocul. **159**:655, 1922.

certainly suggests some endocrine imbalance. No one, however, has offered an explanation of why recurrent hemorrhages of this type occur most frequently in male patients.

Careful analysis of the blood invariably rules out the various blood dyscrasias, such as polycythemia, pernicious anemia, leukemias and primary anemias. The lack of evidence of circulatory or trophic disturbances elsewhere in the body can usually rule out Raynaud's and Buerger's disease [10] in the absence of ophthalmoscopic changes such as have been described by Ballantyne and Michaelson [11] and Cunningham.[12] It must be remembered that, as von Hippel pointed out, "cold feelings in the extremities, paresthesia, etc., are quite commonly found in healthy people and that the pulse of the dorsal artery of the foot is very often not easily palpable. The most definite sign of thrombo-angeiitis is gangrene."

Local phlebitis of the walls of the vessels, as was made clear by Coats many years ago and more recently by Finnoff and Reynolds,[13] Friedenwald [14] and Verhoeff,[15] might cause hemorrhages. The tubercle bacillus has been found occasionally in the peripheral blood vessels of the eye by Otori,[16] Gilbert,[17] Stock [18] and others, so that there can be no doubt as to what causes this phlebitis. In many of the observed cases the hemorrhages were brought about by a diapedesis and were not the result of gross rupture of the walls of the vessels, for when the hemorrhages have absorbed no lesions can be seen. Pathologic examination of such eyes has not been reported to my knowledge.

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10. Marchesani, O.: Eine neue Auffassung des Krankheitsbildes der sogenannten juvenilen rezidivierenden Glaskörperblutungen, Klin. Wchnschr. **13**:993, 1934.

11 Ballantyne, A. J., and Michaelson, I. C.: A Case of Perivasculitis Retinae Associated with Symptoms of Cerebral Disease, Brit. J. Ophth. **21**:22, 1937.

12. Cunningham, J. P.: Retinal Hemorrhages and Exudations in a Young Subject, Tr. Ophth. Soc. U. Kingdom **32**:177, 1912.

13. Finnoff, W. C., and Reynolds, E. M.: Clinical and Experimental Studies of the Spread of Tuberculosis in the Eye, Tr. Am. Acad. Ophth. **38**:152, 1933.

14. Friedenwald, J. S.: Pathology of the Eye, New York, The Macmillan Company, 1929, p. 70.

15. Verhoeff, F. H.: Histologic Observations in a Case of Localized Tuberculosis Chorioretinitis, Arch. Ophth. **1**:63 (Jan.) 1929.

16. Otori, K.: Pathologisch-anatomische mit experimentelle Untersuchungen zur Kenntnis der primären Netzhauttuberkulose, Arch. f. Augenh. **79**:44, 1915.

17. Gilbert, W.: Ueber intra-okuläre Tuberkulose, München. med. Wchnschr. **6**:306, 1914.

18. Stock, W.: Weitere Untersuchungen über hämatogene Tuberkulose der Augen des Kaninchens, Ber. ü. d. Versamml. d. ophth. Gesellsch. **32**:297, 1905; Tuberkulose als Aetiologie der chronischen Entzündungen des Auges, Arch. f. Ophth. **66**:1, 1907.

COMPLICATIONS

The commonest dangers associated with this condition are acute rises in tension, with ultimate loss of the eye; permanent opacities in the vitreous, partially destroying the sight; cataract; detachment of the retina; thrombosis; newly formed blood vessels,[19] vascular veils, and various degrees of proliferating retinitis. Perhaps one of the best descriptions of vascular growths in the vitreous accompanied by hemorrhages is given by Marple.[20] A case similar to one that I am about to present was first described, according to this article, by Coccius in 1859. The patient was a woman aged 36. Marple also cited a similar case in a man aged 27, which was reported by Mauthner and Jaeger (1867). The most interesting thing to me, and something that I have noticed in my last case, is that there is a tendency for these vessels to disappear once the hemorrhages cease, no matter how far forward the vessels may project into the vitreous.

PROGNOSIS

Where vascularization of the vitreous occurs, the prognosis is not necessarily poor. Often the vessels will recede and even disappear if there are no hemorrhages over a period of years. Vascularization of the vitreous has been reported even in older persons, with retention of normal acuity of vision (Flint and Harrington [19a] and Giri [19b]). If the hemorrhages occur solely in the vitreous and there is no accompanying retinitis, the prognosis, though guarded, seems to be fairly good with proper treatment. Whenever the hemorrhages are limited to the retina and there has been an accompanying thrombosis, the prognosis is bad.

TREATMENT

While no one can definitely say that recurrent hemorrhages of nonspecific origin may not occur in the vitreous of young adults, it is the consensus that the hemorrhages are a direct result of a tuberculous process in the blood vessels. Treatment should be more or less empiric and guided by common sense. The importance of fresh air, rest, freedom from overstrain and a diet rich in vitamins has long been recognized as helpful. That tuberculosis of the eye in one form or another may exist without any systemic infection is now generally admitted; however, this conclusion must be derived only after most careful and painstaking clinical and roentgenographic examinations of the patient have been

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19. (*a*) Flint, G., and Harrington, D.: New Vessel Formation in the Vitreous, Brit. J. Ophth. **18**:27, 1934. (*b*) Giri, D. V.: Vascularisation of the Whole Vitreous in a Case of Hemorrhagic Retinitis with Retention of Normal Acuity of Vision, ibid. **18**:24, 1934.

20. Marple, W. B.: Contribution to the Pathology of Vascular Growth into the Vitreous, Tr. Am. Ophth. Soc. **9**:254, 1901.

made, for the treatment will depend somewhat on the results of these examinations. For example, it is not necessary to send a patient with this or any other form of ocular tuberculosis to a sanatorium. The patient should be hospitalized when the hemorrhages are most severe, for it is only in the hospital that adequate local treatment can be given. Local treatment consists of injections of saline solution, atropinization, the use of ethylmorphine hydrochloride and hot applications after clotting of the blood has taken place. Special procedures, such as roentgen irradiation, treatment with ultraviolet rays and autohemotherapy, can also be carried out best while the patient is under careful supervision.

There are many forms of treatment which have been suggested and tried with various measures of success. Perhaps it would be well to mention a few of these—the administration of calcium lactate or calcium gluconate, the intravenous injection of vitamin C (cevitamic acid), the injection of snake venom, the use of the galvanic electric current, the administration of ergot, glyceryl trinitrate, or gold sodium thiosulfate and the injection of foreign proteins and horse serum. One by one these measures may be eliminated for lack of clinical evidence. That tuberculin is helpful in treating other forms of ocular tuberculosis, there can be little doubt. Woods and Randolph,[21] Wilmer,[22] Friedenwald,[23] Gay [24] and Eggston [25] have agreed to this.

REPORT OF CASES

CASE 1.[26]—W. R., a white man aged 28, had his first severe hemorrhage on June 24, 1931. The vision of the right eye with correction was 20/40 and of the left eye 3/200. A thorough survey was made, including a dental examination, roentgenographic examinations of the chest and sinuses, a basal metabolic test, a study of the blood chemistry, a determination of the blood pressure, a complete examination of the blood and urine, a Wassermann test and a general physical examination. The results of all tests and examinations were negative, except that the patient gave a positive reaction to 0.1 cc. of a 1:100,000 dilution of tuberculin and had a slight chronic infection of the right ethmoid sinus and antrum. He was immediately given tuberculin therapy, the injections being con-

21. Woods, A. C., and Randolph, E.: Treatment of Ocular Tuberculosis, Arch. Ophth. **18**:510 (Oct.) 1937. Woods, A. C.: Tuberculosis of the Eye, Internat. Clin. **1**:96, 1933.

22. Wilmer, W. H.: Clinical Aspects of Ocular Tuberculosis, Arch. Ophth. **57**:1, 1928.

23. Friedenwald, J.: Allergy and Immunity in Ocular Tuberculosis, Arch. Ophth. **9**:165 (Feb.) 1933.

24. Gay, L. N.: Treatment of Ocular Tuberculosis with Tuberculin, Arch. Ophth. **3**:259 (March) 1930.

25. Eggston, A. A.: The Use of Tuberculin in Diagnosis and Treatment in Ophthalmology, Arch. Ophth. **8**:671 (Nov.) 1932.

26. This case was reported at the New York Academy of Medicine, Section of Ophthalmology, in April 1935 and a report was published in the ARCHIVES OF OPHTHALMOLOGY (**14**: 860 [Nov.] 1935).

tinued biweekly for two years. In the meantime the patient had seven severe attacks of hemorrhage in the right eye and four in the left eye. During the height of one attack the vision in the right eye was reduced to perception of hand movements at 1 foot (30 cm.) and in the left eye to 2/200. Between attacks, when the fundus could be seen, the veins appeared markedly engorged, and only once was a small retinal hemorrhage seen. The diagnosis of tuberculous perivasculitis was made by numerous observers, though there appeared to be an absence of the usual perivascular inflammatory changes. The patient has had no attacks since July 1932. The vision with correction is 20/15 — in each eye. He is still receiving weekly injections of tuberculin for the purpose of maintaining his acquired immunity, although he has had no hemorrhages for at least five years. No lesion was ever noted in the fundi.

Fig. 1 (case 2).—Right eye, showing newly formed vessels sprouting from the optic disk and retina. The drawing was made three years after the first attack of hemorrhage. The clumps of pigment seen in the nasal quadrant were thought to be of congenital rather than of inflammatory origin. One year after the drawing was made the vessels had completely atrophied and disappeared. The veins are still somewhat dilated and tortuous.

Case 2.—M. S., a white man aged 21, had his first hemorrhage in the left eye in April 1933. The vision has steadily remained blurred in the left eye, whereas the right eye has retained vision of 20/15, in spite of repeated retinal hemorrhages and vascularization of the vitreous. A most exhaustive laboratory study of the patient's blood was made, including determinations of the platelet count, the differential count, the bleeding and clotting times and the calcium, the phosphorus and the cholesterol content and the rest of the usual chemical analyses. All the usual known tests to eliminate tuberculosis and Raynaud's or Buerger's disease were made. Roentgenograms of the sinuses and chest were normal. The

Fig. 2. (case 2).—Left eye, showing preretinal fibrinous tissue and new vessel formation. There is a small streaklike retinal hemorrhage on the nasal side of the disk.

Fig. 3 (case 2).—Drawing of the same eye as shown in figure 2, pictured more anteriorly. The branching of the vessels appears dichotomous, and there are few free terminals. There have been a marked regression of the vessels and a thinning of the fibrinous tissue during the past year.

patient was found to give a slightly positive reaction to 0.01 mg. of tuberculin and treatments were begun several months before he came under my observation. The injections of tuberculin were continued throughout the course of the disease, and the patient is still receiving weekly injections. Because of a low basal metabolic rate, —20, the patient has been taking small doses of thyroid. When the basal metabolic test was repeated, the rate was found to be —5, and the administration of thyroid was discontinued. At one time it was thought the patient had a low platelet count. On repeated checks, it was found to be normal. In order to build up the patient's resistance, numerous therapeutic measures were used. He was given a large number of intravenous injections of cevitamic acid and five injections of gold sodium thiosulfate. He was told to lead an outdoor life, with a moderate amount of exercise, and to stay in bed after any severe hemorrhage, until the vision showed a definite return to normal. The most interesting feature of the case appears to be the atrophy or recession of the blood vessels, especially noted in the right eye. The small vessels projecting from the disk and the whorl arrangement of blood vessels shown in figure 1 have entirely disappeared. The only sign that there has been any active hemorrhage is some scar tissue with slight clumping of the retinal pigment. There seems also to be atrophy of the fibrinous tissue of the left eye, with great improvement in the visual field, which at one time was completely blotted out and could be taken only with the ophthalmoscope light. The patient's vision on his last visit, March 1938, was 20/15 in the right eye with a —0.75 D. sph. and 20/50 in the left eye with a —1.50 D. sph.

COMMENT AND SUMMARY

The striking difference in the picture of the fundi in these two cases of recurrent hemorrhages in the vitreous deserves some comment. Both patients, young men in apparent good health, were observed for several years, during which time there was marked disturbance of vision and at times blindness was threatened because of the severity of the hemorrhages. That both patients should recover, one with normal vision in each eye and the other (case 2) with restoration of two thirds of his sight in spite of a partially vascularized vitreous, should be of more than passing interest. A diagnosis of tuberculous perivasculitis could not be made with certainty in case 1, as at no time were there visible changes in the vessels. Most observers agreed that the veins appeared markedly engorged, especially at the time or just previous to an attack of hemorrhage. At the end of six years, long after the hemorrhages had ceased, both fundi appeared normal. The veins still looked somewhat dilated, and there were no scars or other signs of an old inflammatory process in the retina. The vitreous, except for a few filamentous opacities, had also cleared, and the patient experienced no ocular discomfort. The faint opacities in the posterior cortex of the lens have not increased in density during the past five years and have caused no measurable loss of vision.

The hemorrhages in the vitreous in case 2 illustrate another type and undoubtedly are not the same as those first described by Eales, though the etiologic basis may be the same. The condition in this case resembles in many ways that in the 3 cases recently reported by Knapp [27] under the heading "Formation of Preretinal Connective Tissue in the Vitreous in Acute Choroiditis."

27. Knapp, A.: Formation of Preretinal Connective Tissue in the Vitreous in Acute Choroiditis, Arch. Ophth. **18**:558 (Oct.) 1937.

AN OPHTHALMIC CARRIAGE

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Though ophthalmology as a medical science has ever marched in the forefront, there are certain portions of clinical work wherein it seems to lag behind. A review of these deficiencies and of possible methods for their correction demonstrated an urgent need for improvement in bedside facilities for the study of patients with ophthalmic conditions.

A portable instrument carriage seemed to me essential to facilitate study in cases in which ophthalmic consultation is required and for the transportation of equipment during rounds in the hospital.

If one reviews the bedside notes made during such rounds, it becomes evident that in many instances there has been an omission of study procedures owing to a lack of satisfactory facilities. The omission of a postoperative test of the visual acuity of the sound eye as well as of the affected eye is not uncommon; and when the visual acuity is studied, the conditions under which the test is made are ordinarily far inferior to those under which it was made on the same patient previous to admission to the hospital. This is but one example of the need for improved bedside facilities for ophthalmologic study, and it was determined to endeavor to provide the means to supply this need.

A description of the portable carriage devised follows:

An oak filing cabinet of standard type was altered so that its final exterior dimensions were height, 26 inches (66 cm.); depth, 27¾ inches (70 cm.), and width, 16½ inches (42 cm.). This alteration provided for two large drawers, which open from the front end of the carriage. They were shortened to provide a space within the cabinet for the storage of eight dry cells (ignition type, used ordinarily for the operation of doorbells and buzzers), for the accommodation of a rheostat (for the reduction of house current for the hammer lamp) and an automatic reel for the lamp cord and for the housing of charts for testing visual acuity. Further space was allowed for a small rheostat for the reduction of the current of the aforementioned batteries and an incorporated jack to receive a plug (so that electric ophthalmoscopes, retinoscopes, an O'Brien slit lamp, a transilluminator and other devices can be operated from the battery supply).

From the Department of Ophthalmology, Long Island College of Medicine.

On the top of this cabinet was mounted a brass tray having an upturned flange of 1½ inches (3.7 cm.). At one end of this tray a slot with a surrounding flange was provided, so proportioned that two standard charts for testing visual acuity can be raised and lowered with their appropriate frames and locking mechanism. At the opposite ends of the cabinet, in each corner, were provided two tubular sockets proportioned for the reception of a rod and adapted with lock nuts, so that the rod can be elevated or depressed and fixed in position as required. On the upper end of this rod a hammer lamp was mounted in a lockable universal joint. (This permits the hammer lamp to be

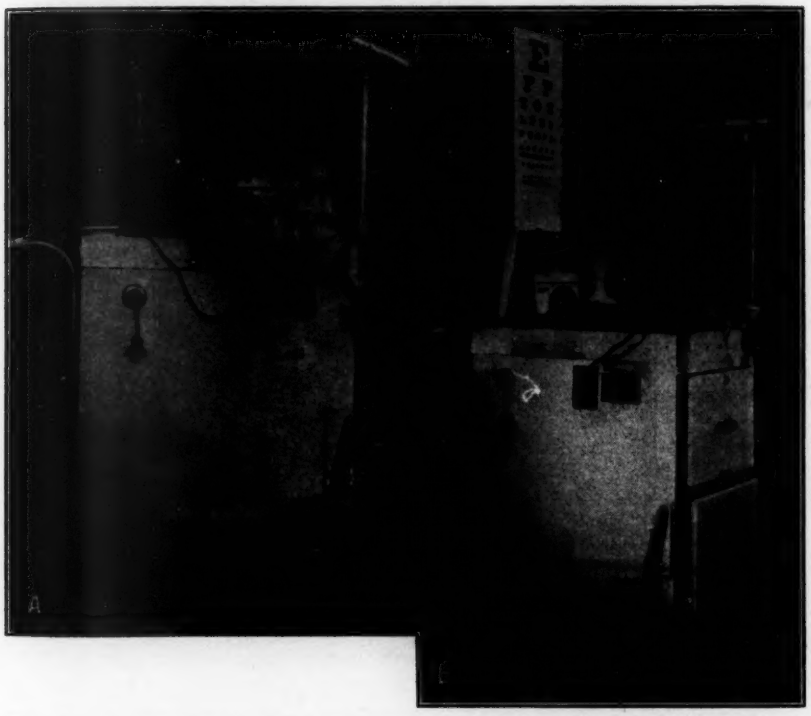

Fig. 1.—*A,* ophthalmic carriage arranged for the physician when doing bedside dressings. The solution bottle, dressing jar, treatment boxes and adhesive tape carrier are in evidence. For purposes of illustration, the O'Brien hand slit lamp and binocular magnifier are in view. *B,* ophthalmic carriage showing the arrangement of the light and the visual acuity test chart. It will be noted that the chart is so elevated that it can be seen by the patient without material elevation of the head of the bed.

directed for the illumination of a visual acuity test chart, the dressing tray or the examination field of a patient. It also permits its use for indirect ophthalmoscopic and retinoscopic examinations and for oblique illumination. A ground glass disk was devised for the hammer lamp, to soften and diffuse the light, and an adjustable diaphragm was also

provided, so that the hammer lamp can be used as a muscle light or for other purposes.) The hammer lamp is thus readily demountable, so that it can be used as a hand instrument and can be stored with its accessories in a rack provided for that purpose in the upper drawer of the cabinet. The remaining space in the upper and lower drawers of the cabinet was equipped with the aforementioned instruments and accessories as well as with a hand perimeter, a folding campimeter, a small trial case with frames and accessories, a visual acuity test chart for testing near vision, a Placido disk, an ophthalmoscope handle with adaptors to receive various makes of heads, Stilling's pseudoisochromatic

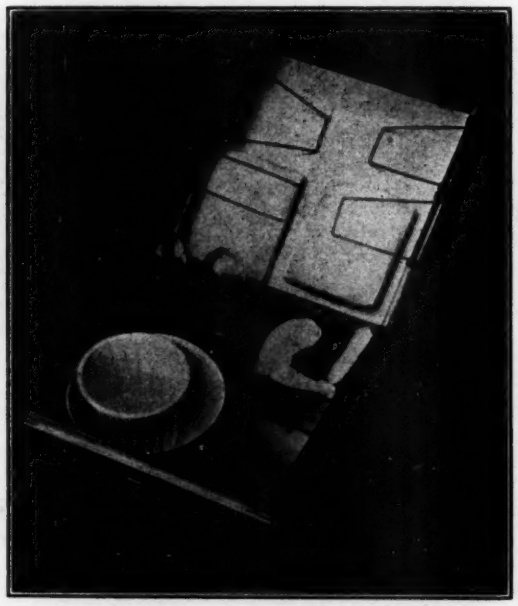

Fig. 2.—Individual dressing box. This box is equipped and assigned to each patient as he enters the hospital. The body of the box is so divided that there is room for four medication bottles. These bottles have glass stoppers which are ground to the outside of the neck of the bottle. The graniteware cup is merely used as a container for the sterile paper cup, the latter being put into the box just before the dressing is commenced. The clips in the lid of the box retain dressing pads, hard sponges, soft sponges, loosely and tightly wound cotton applicators, all sterile-wrapped. The loose-wound applicators are used for instilling the medication.

plates for testing color sense; a combination millimeter-inch measuring tape, Berens' combination accommodation rule and one set of muscle prisms.

The upper surface of the cabinet was covered with a heavy brown linoleum, which was so sealed in place as to be effective in waterproofing the top of the cabinet. The top, with its brass rim, thus provides sufficient space for six individual dressing boxes. (Each patient

is allotted a special cadmium-plated dressing box when he is admitted to the hospital. It contains special bottles of such freshly prepared medication as is ordered for him, together with applicators, minor dressings and a cup for saline solution, an adhesive tape dispenser [the new transparent tissue is being used] and flasks of sterile saline solution, sponges and dressings.)

The carriage was mounted on pneumatic tires and a swivel wheel was provided with a brake mechanism. There were added a convenient handle for propelling the vehicle and a fender bar to protect door frames.

A study of figure I will make the description sufficiently complete.

The following equipment is kept on or in the carriage:

1. Zeiss hammer lamp, with attached rheostat, wiring and wire spool assembled
2. Diffusing lens attachment for the hammer lamp
3. Selective aperture diaphragm attachment for the hammer lamp
4. Hand condensing lens, for use with the hammer lamp
5. Six cadmium-plated dressing boxes
6. Three glass-capped medication bottles in each box
7. Six packages of toothpick applicators (sterile-wrapped) in each dressing box
8. Six packages of dressings for the eye (sterile-wrapped) in each dressing box
9. Three packages of cotton balls (sterile-wrapped) in each dressing box
10. One enamelware container for a sterile paper cup
11. One roll of cellulose adhesive tape and dispenser (table type)
12. Two American Medical Association visual acuity test charts in adjustable holders
13. Visual acuity test charts for near vision (two forms)
14. One O'Brien hand slit lamp
15. One Berger loupe
16. One small trial case, with lenses and accessories
17. One adjustable trial frame
18. One red glass in a holder (for the study of muscles)
19. One transilluminator (Zeiss)
20. One electric retinoscope (L.I.C.H.)[1]
21. One accommodation rule (Berens' combination)
22. One vest pocket dark room
23. One ophthalmoscope handle, flexible cord and jack plug (assembled)
24. One hand perimeter (Schweigger's)
25. One hand campimeter (Peter's)
26. One Placido disk
27. Plates for testing color sense (Ishahara's test)
28. One millimeter-inch tape (meter long)
29. Instrument for Maddox wing test
30. Eight dry cell batteries; one spring reel for the lamp cord, with a receptacle plug (assembled for the current in use)
31. One hand pump for the inflation of tires
32. One extra bulb for the hammer lamp

The firm of J. H. Penny cooperated in the construction of this carriage.

1. This instrument was devised by the department of ophthalmology, Long Island College Hospital.

TOPOGRAPHY AND FREQUENCY OF COMPLICATIONS OF UVEAL SARCOMA

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From 1928 to 1935 inclusive, 995 eyeballs were examined at the Eno Laboratory of the New York Eye and Ear Infirmary. Of this number, 126 (12.8 per cent) were found to have sarcoma of the uvea. In addition to these tumors, 62 other uveal tumors the histologic sections of which were available were studied. This material constitutes the basis of this report.

The chief purpose of this paper is to present the results of a study of this series of uveal tumors from the standpoint of topography and to discuss the frequency of the complications arising from such growths. Other interesting information which was correlated in studying the sections is also reported.

TABLE 1.—*Classification of Uveal Tumors*

Site	Present Series	Fuchs	Lawford and Collins
Sarcoma of choroid alone	164 (88%)	221 (85%)	94 (91%)
Sarcoma of choroid and ciliary body	2 (1%)	...	2 (2%)
Sarcoma of ciliary body	4 (2%)	22 (9%)	6 (6%)
Sarcoma of ciliary body and iris	9 (4.5%)	...	..
Sarcoma of iris	9 (4.5%)	16 (6%)	1 (1%)

Table 1 shows how the tumors in this series were grouped and how this classification compared with that of other authors. Fuchs [1] collated all previous papers on sarcoma of the uvea and added 22 cases of his own to those already reported, making a total of 259. Lawford and Collins [2] reviewed the reports of 103 cases.

In connection with the data in table 1, it must be stated that the point of origin of the tumor which extended over more than one part of the uvea was extremely difficult to determine.

SARCOMA OF CHOROID

For purposes of study, the tumors of the choroid alone and of the choroid with extension to the ciliary body (166) were divided on the following bases:

Site.—The choroid is commonly divided into an anterior, an equatorial and a posterior zone. I have subdivided these zones, in turn, into

1. Fuchs, E.: Das Sarkom des Uvealtractus, Vienna, Wilhelm Braumüller, 1882.

2. Lawford, J. B., and Collins, E. T.: Ophth. Hosp. Rep. London **13**:12, 1891.

a nasal and a temporal region and these again into a superior and an inferior region. Although tumors of the circumpapillary and macular regions are located posteriorly, they were classified separately, as is shown in table 2.

As can be seen from table 2, of 166 sarcomas of the choroid, 86 (51.7 per cent) were located posteriorly in contrast to 33 (19.8 per cent) located anteriorly and 27 (16.2 per cent) located equatorially. The largest individual sector was the posterior temporal region, where 41 (24.7 per cent) of the choroidal sarcomas were found, whereas only 8 (4.8 per cent) were located posteriorly and nasally. The total number of tumors in the temporal zone, that is, in all sections of this zone amounted to 62 (37.3 per cent), while the total number of tumors

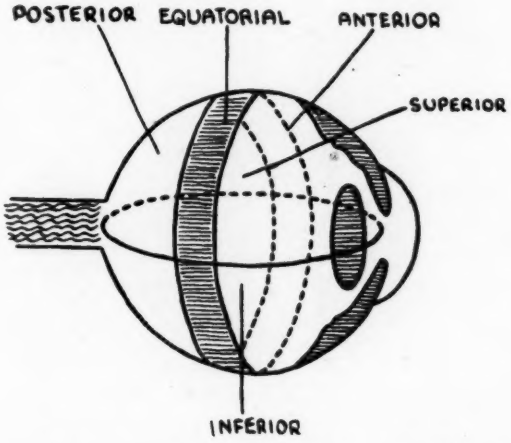

Diagram illustrating the division of the eyeball into zones.

in the nasal region amounted to 23 (13.8 per cent). It is of interest to note that 14 tumors were located in the circumpapillary region and were beginning to encroach on the disk. Forty-seven tumors (28.2 per cent) could not be localized temporally or nasally. The foregoing material, however, is sufficient to justify the conclusion that most sarcomas of the choroid arise posteriorly and especially temporally. This is, of course, fortunate since central vision is affected early and attention is drawn to the eye.

In table 3 is presented a comparison of my findings in regard to location with those of Fuchs [1] and of Lagrange,[3] as given by Lagrange's pupil Baudouin. The data for my series are more conclusive because of the greater number of localizations ascertained.

3. Lagrange, F.: Traité des tumeurs de l'oeil, Paris, G. Steinheil, 1901.

TABLE 2.—*Topographic Distribution of Sarcoma of the Choroid* *

Site	T	TS	TI	Mac	Total No.	Total Percentage	N	NS	NI	Total No.	Total Percentage	C No.	C Percentage	Und. No.	Und Percentage	Entire Series: Total No.	Entire Series: Total Percentage
Anterior segment	9	1	1	..	11	6.6	8	1	1	10	6.0	..	...	12	7.2	33	19.8
Equatorial segment	7	0	3	..	10	6.0	5	0	0	5	3.0	..	...	12	7.2	27	16.2
Posterior segment	20	7	7	7	41	24.7	7	0	1	8	4.8	14	8.4	23	13.8	86	51.7
Whole globe	..	..	..	..	..	...	..	..	..	..	...	..	...	..	...	8	4.9
Half globe	..	..	..	..	..	...	..	..	..	..	...	..	...	..	...	8	4.9
Location unknown	..	..	..	..	..	...	..	..	..	..	...	..	...	..	...	4	2.5
Total	..	..	..	..	62	37.3	..	..	..	23	13.8	14	8.4	47	28.2	166	100

* T indicates the temporal region; TS, the temporal superior region; TI, the temporal inferior region; Mac, the macular region; N, the nasal region; NS, the nasal superior region; NI, the nasal inferior region, and C, the circumpapillary region. Und indicates sarcomas, the location of which was undetermined.

Age.—The average age of the patients with sarcoma of the choroid in the present series was found to be 52.6 years, the youngest patient being 14 and the oldest, 84. Fuchs found the average age of 259 patients with uveal sarcoma to be 42.2 years. Lawford and Collins ascertained it to be 48.42 years, and Pawel [4] gave the average age as 48.7 years.

Sex.—The distribution of the tumors according to sex is presented in table 4. A comparison of my findings with those of other authors is made. As can be seen, the tumors occurred equally in the two sexes.

TABLE 3.—*Comparison of the Distribution of Tumors in the Present Series with That Reported by Other Authors*

Site	Present Series	Fuchs	Lagrange
Anterior segment	33 (19.8%)	14	38
Equatorial segment	27 (16.2%)	12	7
Posterior segment	86 (51.7%)	74	24
Whole globe	8 (4.9%)	..	..
Half globe	8 (4.9%)	..	..
Unknown	4 (2.5%)	121	61

TABLE 4.—*Distribution of Tumors on the Basis of Sex*

Sex	Present Series	Fuchs	Lawford and Collins	Pawel
Male	54 (36.4%)	137 (53%)	59 (56%)	47 (47%)
Female	44 (25%)	166 (44.7%)	44 (44%)	53 (53%)
Unknown	68 (38.6%)	6 (2.3%)		

TABLE 5.—*Distribution of Tumors on the Basis of the Eye Affected*

Site	Present Series	Fuchs	Lawford and Collins	Pawel
Right eye	39 (23.4%)	108	41	43
Left eye	36 (21.6%)	101	60	48
Bilateral	..	5	..	..
Unknown	91 (55%)	45	2	9

Eye Affected.—The data found by other authors with regard to the eye affected are present in table 5 for comparison with similar data for the present series.

Pigmentation.—At one time sarcomas of the choroid were divided into pigmented and nonpigmented growths, the latter being the leukosarcomas. The opinion that all tumors possess a prepigment substance which is converted into pigment by oxidation, as expressed by Samuels,[5] has gained ground. This conversion varies according to the circulatory supply. Thus, some tumors are pigmented in some areas and

4. Pawel: Arch. f. Ophth. **49**:1, 1900.
5. Samuels, B.: Pennsylvania M. J. **36**:895, 1933.

not in others. If one examines sufficient sections, it will be found that practically all tumors are pigmented. The findings in the present series of tumors in regard to pigmentation are presented in table 6 and are compared with those of Fuchs [1] and Pawel.[4] All available sections of the 33 nonpigmented sarcomas were examined, but the evidence is not conclusive, since the entire tumor was never sectioned. If other sections had been taken, pigment might have been found. However, the predominance of pigmented tumors is apparent from the figures given.

Types of Cells.—The types of cells found in the sarcomas were either round, spindle or mixed. I found, however, that a sharp demarcation between the types of cells is not possible. The same cell, when sectioned at a different angle, may have a different appearance. Round cells were found in 47 of the 166 tumors in this series, spindle cell in 54 and mixed round and spindle cell in 41. The type of cells was not determined in the remainder of the growths. Fuchs reported that 65 of 147 sarcomas of the choroid were of the spindle cell type, 40 were of the round cell type and 28 were of the mixed round and spindle cell type; the remainder were of various types.

TABLE 6.—*Distribution of Tumors on the Basis of Pigmentation*

Type	Present Series	Fuchs	Pawel
Pigmented	127 (76.5%)	229 (88.4%)	46 (78%)
Nonpigmented	33 (19.9%)	30 (11.6%)	13 (22%)
Unknown	6 (3.6%)		

Shape.—No definite figures were given by any of the foregoing authors as to the shape of the tumors. Yet the determination of the shape is valuable for prognosis, since an early tumor is inclined to be flat because it is compressed by the lamina vitrea on one side and by the sclera on the other. The tumor then grows in the perichoroidal space without meeting much resistance until it reaches the attachment of the ciliary body anteriorly or the zone in which the choroid is bound to the sclera posteriorly. At this stage the tumor has the shape of a loaf of bread. The tumor continues to increase in size and since the lamina vitrea offers the least resistance, it breaks through at some point, forming a hole, and the subsequent growth results in a mushroom-shaped tumor. Further growth results in a spherical mass, which eventually occupies a lesser or a greater portion of the eyeball. The distribution of the 166 tumors in regard to shape is presented in table 7.

COMPLICATIONS

The following complications may be encountered in cases of uveal tumor.

Detachment of the Retina.—The retina immediately surrounding the growth is not necessarily detached from the underlying choroid. In

some cases it remains attached to the head of the tumor. In most cases, however, albuminous fluid collects underneath the retina which, according to Parsons,[6] is a result of the irritation set up in the choroid by the tumor and, according to Samuels,[5] is a result of transudation from the choroidal vessels due to a disturbance of the circulation by the growing mass. Frequently, however, a retinal detachment also arises in other parts of the retina because of the sinking of the fluid. Sometimes a large detachment develops and hides the tumor behind it, making diagnosis somewhat difficult.

In the present series, detachment occurred in 127 cases; it did not take place in 37 cases, and in 2 the status of detachment was not determined.

It is of interest to note that sarcomas of the circumpapillary region almost always cause detachment of the retina. Of 14 such tumors, 13 (92.8 per cent) were complicated by detachment. The frequency of the occurrence of detachment was about the same for all the other tumors.

TABLE 7.—*Distribution of Tumors on the Basis of Shape*

Shape	Number	Percentage
Flat	13	7.8
Loaf of bread	27	16.3
Spherical	27	16.3
Mushroom shaped	61	36.7
Irregular	16	9.6
Whole globe	8	4.8
Half globe	5	3.0
Unknown	9	5.5

Glaucoma—Glaucoma is a typical outcome of uveal tumor and is considered to mark the second stage in the development of the growth. Eighty-two of the 166 eyes were glaucomatous, 89 were nonglaucomatous and the condition of 4 with reference to glaucoma was not determined. The glaucoma is not directly due to the new growth but is caused by the pressure of the subretinal fluid, which presses forward on the lens and the iris. Since frequently in these cases no preoperative histories were available, the criteria of glaucoma in the microscopic sections were an occluded anterior iris angle, patches of degeneration in the iris and abnormal excavation of the disk with atrophic changes of the nerve fibers.

In regard to the relation of the topographic localization of the tumors and the frequency of glaucoma as a complication, it was noted that they were about equal (table 8).

6. Parsons, J. H.: Pathology of the Eye, London, Hodder & Stoughton, 1905, vol. 2.

Extraocular Extensions.—Extraocular extensions of the growth take place along the perforating vessels. From here they continue to grow into the orbit, and at the same time they may also metastasize to other parts of the body. In the present series of tumors the presence of tumor cells in the perforating vessels was considered as evidence of extraocular extensions. Thus, I found that 60 (36.1 per cent) of the tumors showed extraocular extensions, while 102 (61.4 per cent) did not. The status of 4 (2.5 per cent) of the tumors in regard to these extensions was not determined.

The site of the tumor did not seem to have any influence on the frequency of extraocular extensions (table 9).

Necrosis.—Another possible outcome of choroidal sarcoma is necrosis of the tumor tissue. In some cases of sarcoma a severe iridocyclitis breaks out. Anatomically, this has been found to be due to

TABLE 8.—*Relation of Topographic Localization and the Presence of Glaucoma*

Segment	Glaucoma	No Glaucoma
Anterior	16 (53.3%)	14 (46.7%)
Equatorial	13 (48.1%)	14 (51.9%)
Posterior	39 (45.4%)	47 (54.6%)

TABLE 9.—*Data on Extraocular Extensions in Relation to Distribution of Tumors*

Segment	Extraocular Extensions	No Extraocular Extensions
Anterior	12 (40%)	18 (60%)
Equatorial	8 (29.7%)	19 (70.3%)
Posterior	27 (32.4%)	59 (68.6%)

toxins resulting from the tumor. Deficient nutrition due to poor circulation of the blood is the cause of the necrosis. Samuels,[7] in his paper on "Anatomic and Clinical Manifestations of Necrosis in Eighty-Four Cases of Choroidal Sarcoma," amply and fully described this result. He stated that of 106 sarcomatous eyes, 31 were highly necrotic. In only a small percentage of these was necrosis suspected preoperatively. Twenty of 31 eyes were enucleated under other diagnoses. No studies of necrosis were made in this series, and nothing further was added to Samuels' conclusions.

In studying the present series of tumors it was found to include 10 metastatic carcinomas of the choroid the primary origin of which could not be ascertained. Histologically, 6 of them were adenocarcinomas and 4 were medullary carcinomas. Four were in the circumpapillary region, and 4 occupied the posterior part of the eye. In 2

7. Samuels, B.: Anatomic and Clinical Manifestations of Necrosis in Eighty-Four Cases of Choroidal Sarcomas, Arch. Ophth. **11**:998 (June) 1934.

cases the original site could not be located because the tumor occupied the entire globe. The posterior location of these tumors indicated that the metastases were arrested in the larger vessels of the choroid.

SARCOMA OF THE CILIARY BODY

In this series of tumors there were 4 (2 per cent) sarcomas of the ciliary body alone and 9 (4.5 per cent) of the ciliary body with extension to the iris. Fuchs found the percentage to be the same in his series. It was difficult to determine definitely the site of origin of the sarcomas of the ciliary body and iris and to state how many extended from the ciliary body to the iris or vice versa. It seemed most probable that the place of origin was the ciliary body. Histologically, all tumors of this group were found to be mixed round and spindle cell sarcomas. Of the total number of 13 tumors, only 1 was not pigmented. The average age of the patients in this group was 50.1 years.

SARCOMA OF THE IRIS

In this series there were 9 (4.5 per cent) sarcomas of the iris. Histologically, they were mixed sarcomas. All 9 were pigmented. The average age of the patients was 40.3 years. Fuchs [1] in his series found the average age to be 31 years. The average age of persons with sarcoma of the iris has shown itself to be much lower than that of persons with sarcoma of the choroid.

SUMMARY AND CONCLUSIONS

In regard to the topographic distribution of choroidal sarcomas, it was found that most tumors are localized posteriorly and especially temporally.

In the present series 12.8 per cent of all enucleated eyes were removed because of uveal sarcomas.

Eighty-eight per cent of all uveal sarcomas are located in the choroid.

The average age of persons with sarcoma of the choroid was found to be 52.6 years.

Sarcomas of the choroid are distributed equally in the two sexes.

Most choroidal tumors (76.5 per cent) are pigmented.

The spindle cell sarcomas predominate over the round cell type.

The most common choroidal sarcomas on the basis of shape are those shaped like a loaf of bread and a mushroom.

Detachment of the retina occurs in more than 75 per cent of all cases of choroidal sarcoma.

Almost all circumpapillary sarcomas cause detachment of the retina (92.8 per cent).

One half of the eyes enucleated for sarcoma showed glaucomatous changes. The topographic distribution does not influence the frequency of glaucoma.

Extraocular extensions were found in 36.1 per cent of all cases; in 61.4 per cent there were no extensions. The site of the sarcoma has no bearing on the frequency of extensions.

Metastatic tumors of the choroid appear principally in the posterior segments.

Sarcomas of the ciliary body frequently involve the iris. They are generally nonpigmented.

The average age of patients with sarcoma of the iris is lower than that of patients with sarcoma of the choroid, that is, 40.3 years.

INOSITOL IN THE OCULAR TISSUES

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The knowledge of the chemical composition of most tissues is yet unsatisfactory. A few organs have been studied intensively, and their composition is relatively well known; on the contrary, for many other organs systematic data are still lacking. To undertake a study of physiologic chemistry before having a sufficient knowledge of "anatomic chemistry" results in incomplete or even false interpretations.

The analyses of the ocular tissues are fairly complete. This is particularly true for the lens. For 100 Gm. of lens, the composition of about 98.5 Gm. is known. The remaining 1.5 Gm. contains substances which, acting as catalysts, play an important role in spite of their low concentrations. Other substances enter the lens by diffusion; they do not act as metabolites, and their role is unimportant. But although the unknown fraction of the lens is a small part of the total weight, investigation of it may reveal the presence of substances reaching high concentrations, even higher than that of dextrose. Inositol is such a substance and is the subject of this article. This investigation, however, has not been limited to the lens but has been extended to all ocular tissues.

Inositol (cyclohexanhexol) was discovered in 1850 by Scherer. Because of its sweet taste, this substance was for a long time considered as a sugar and was called inosite or *Muskelzucker*. It is known to be widely distributed in both animal and plant tissues. A great interest arose in this substance when it was discovered that phytin was the phosphoric ester of inositol and that the enzyme, phytase, occurred in many animal tissues. Later, inositol was found in the urine of persons with diabetes insipidus and diabetes mellitus. The discovery of inosituria resulted in contradictory interpretations. It is actually well demonstrated that any polyuria, with or without glycosuria, is accompanied by inosituria. Inosituria is a consequence of the polyuria, not of the glycosuria.

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The role of inositol in the tissues, although still actually unknown, seems to be important in the biologic processes. Its presence and its distribution in the ocular tissues have not yet been the subject of experimental investigation. For this reason this work has been undertaken.

EXPERIMENTAL PROCEDURE

Normal eyes from 3 or 4 year old cattle were immediately removed after slaughter of the animal; dissection of the ocular tissues was started as soon as possible. An unavoidable delay of a few hours was, however, necessary before the extraction was begun. Since inositol is stable, its destruction was unlikely; on the contrary, a slight increase arising from postmortem changes is possible.[1]

The method of Young [2] for the determination of inositol was used. Samples of corneal epithelium and conjunctiva weighing from 20 to 30 Gm., of vitreous weighing from 500 to 1,000 Gm. and of other tissues weighing from 60 to 150 Gm. were used.

The samples were extracted during two periods of twenty-four hours with a 70 per cent aqueous solution of acetone. For the first extraction, sufficient acetone was added to make a 70 per cent aqueous solution, depending on the amount of water in the tissue.

The acetone of the extract was removed by evaporation on the water bath. The remaining aqueous solution was extracted twice with ether and then evaporated to 100 cc. on the water bath. Extraction with acetone and ether gives better results than hydrolysis with hot alkali because it is more specific. All the tissues were extracted in this manner with the exception of the vitreous humor, which was desiccated before extraction.

Many interfering substances were removed by precipitation with mercuric sulfate in acid solution. After neutralization, the solution was filtered. The mercury was removed by means of hydrogen sulfide and filtration.

Barium hydroxide and 95 per cent ethyl alcohol were used to precipitate inositol. The precipitate was separated by centrifugation and was hydrolyzed by heating after acidification. The barium was removed by filtration, and the clear filtrate was reduced to a small volume. The inositol was precipitated by means of acetone and ether.

The precipitate formed under these conditions was not pure inositol. A certain amount of salts passed during these different procedures, depending on the tissue. The percentage of inositol was determined by a method of titration which is based on a reduction of potassium iodomercurate in alkaline solution. For this titration, an aliquot of solution of the precipitate containing 2 to 5 mg. of inositol was used. After the addition of sulfuric acid, an excess of iodine was added and titrated with thiosulfate.

RESULTS

The accompanying table shows the distribution of inositol in ocular tissues.

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1. Winter, L. B.: Inositol Metabolism in the Mammalian Heart, Biochem. J. **28**:6, 1934.

2. Young, L.: Determination of Inositol in Animal Tissues, Biochem. J. **28**: 1435, 1934.

The high concentrations of inositol in the lens and in the optic nerve permitted the isolation of the substance in almost pure form. The inositol obtained from the lens at the second crystallization had a melting point of 220.5 C. (uncorrected); that of the optic nerve had a melting point of 222 C. (uncorrected). The melting point of a commercial inositol was 223 C. (uncorrected). The inositol isolated from the lens was optically inactive like that isolated from the brain.[3]

The amount of inositol in the vitreous humor was small. As a rule, the figures obtained by titration were lower than 2 mg. in 100 Gm. of vitreous humor. It was questionable whether this slight reduction was due to inositol or to compounds liberated during the procedure. An attempt to isolate it was unsuccessful. Inositol in the vitreous humor, if present, was probably a product of diffusion and not a metabolite.

Inositol in Ocular Tissues in Milligrams per Hundred Grams of Tissue

Whole Cornea		Corneal Epithelium		Corneal Stroma		Conjunctiva		Iris	
Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry
8.0	42.9	26.5	132.5	5.3	28.2	6.05	33.4	22.0	118.0
6.5	34.8	17.2	86.6	5.1	27.2			27.5	147.5
		15.9	76.6					35.0	187.5

Lens		Vitreous Humor		Retina		Choroid*		Sclera		Optic Nerve	
Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry
150	395	Traces	—	13.4	107.5	35.5	206.8	14.0	50.5	103	342
172	450	Traces	—	16.6	133.0	32.5	187.8	10.5	37.8	96	321
154	405	Traces	—	17.0	136.2	29.2	168.5	13.5	48.6	89	298
126	330	Traces	—	14.5	116.3					111	372
177	465	Traces	—							90.5	303
149	392										

* The retinal pigment epithelium was removed with the choroid.

COMMENT

An excellent survey of the literature up to 1926 has been made by Needham.[4] In spite of rather large amounts of information, knowledge concerning metabolism of inositol is still scanty.

Since inositol is widely distributed in animals and plant tissues, the amount daily ingested in the food is relatively high. However, inositol as such is destroyed by the intestinal bacteria, and only a small fraction is absorbed through the intestinal walls. The inositol is likely absorbed in the form of phytin.

3. Momose, G.: On the Inositol of Brain and Its Preparation, Biochem. J. **10**:120, 1916.

4. Needham, J.: Die physiologische Bedeutung der Cyclosen, Ergebn. d. Physiol. **25**:1, 1926.

Inositol in the blood reaches only low concentrations.[5] The concentrations in tissue are much higher and cannot be explained by simple diffusion. From beef brain, Thudichum[6] has isolated 10 Gm. of pure inositol from 50 pounds (22,679 Gm.) of tissue, or 44 mg. per hundred grams. For human brain, the figures given by the same author are higher: 193 mg. per hundred grams in the gray matter and 217.2 mg. per hundred grams in the white matter. Since this early publication, the data found in the literature show a wide range of variation depending on the method used and the experimental conditions. According to recent determinations, inositol can reach concentrations higher than 170 mg. per hundred grams in the brain. About 85 mg. per hundred grams is found in fresh heart muscle, and about 125 mg. per hundred grams is found in the same tissue some hours after death. The amount of inositol in skeletal muscle seems to be much lower.[7]

Furthermore, previous investigations show that the tissues are able to synthesize inositol, for neither suppression of inositol from the food for a long period nor polyuria with inosituria is able to decrease the amount of inositol in the animal tissues. Since the injection of dextrose increases the amount of inositol in the incubating egg and since the injection of insulin decreases the sugar in the blood and increases the inositol in the tissues, it may be supposed that dextrose plays a role in the synthesis of inositol.[8]

The question arises as to the nature of the inositol in the tissues. Since the concentration of inositol increases with the autolysis, the hypothesis of a precursor of inositol, inositogen, has been suggested.[9] It is possible that the inositol itself is partly free and partly bound with a molecule containing phosphorus. The present methods do not allow more than an estimation of these two fractions.[1]

The final fate of inositol is unknown. It has been suggested that inositol is changed into glycogen, dextrose or perhaps into lactic acid in the vertebrate cardiac muscle.[10] However, inositol in the food does

5. Needham, J.: Studies on Inositol: II. The Synthesis of Inositol in the Animal Body, Biochem. J. **18**:891, 1924.

6. Thudichum, J. L. W.: Die chemische Konstitution des Gehirns des Menschen und der Tiere, Tübingen, Franz Pietzcker, 1901, pp. 40 and 276-278.

7. Gregory, R. A.: A Modification of Young's Method for Determination of Inositol in Animal Tissues, Biochem. J. **29**:2798, 1935. Winter.[1] Young.[2]

8 (*a*) Needham, J.: Studies on Inositol: III. The Metabolic Behaviour of I-Inositol in the Developing Avian Egg, Biochem. J. **18**:1371, 1924. (*b*) Needham, J.; Smith, W., and Winter, L. B.: Insulin and Inositol, J. Physiol. **57**:lxxxii, 1923.

9. (*a*) Rosenberger, F.: Weitere Untersuchungen über Inosit, Ztschr. f. physiol. Chem. **64**:341, 1910. (*b*) Needham, J.: Studies on Inositol: I. A Method of Quantitative Estimation, Biochem. J. **17**:422, 1923.

10. Boyland, E.: Chemical Changes in Muscle: II. Invertebrate Muscle; III. Vertebrate Cardiac Muscle, Biochem. J. **22**:362, 1928.

not increase the respiratory quotient,[11] and perfusion of tissue with inositol gives no formation of dextrose or lactic acid.[12] The experiments done with the Warburg apparatus are not consistent.[13]

It has been suggested that inositol has no other role than that of bringing phosphorus into the organism.[14] If this were true, why does the organism show an active synthesis of inositol and why is there a constant concentration of inositol in the tissue when the diet is free from inositol and or when there is prolonged polyuria with inosituria? Hypotheses on the subject will not add anything more, for experimental facts are missing.

The results of the investigations show that the concentration of inositol varies greatly with the type of ocular tissues. It is high in the lens and in the optic nerve. Further experimental work will perhaps give a clue to the relation of metabolism to these high concentrations.

CONCLUSIONS

Inositol was found in each of the bovine ocular tissues, namely: conjunctiva, corneal epithelium, corneal stroma, iris, lens, retina, choroid, sclera and optic nerve. The presence of inositol in the vitreous humor is questionable.

Its concentration is relatively constant in a definite type of tissue but varies broadly in different tissues. The concentration is remarkably high in the lens (150 mg. per hundred grams of wet tissue) and in the optic nerve (100 mg. per hundred grams of wet tissue).

The role played by inositol in these tissues is still unknown.

To consider inositol as a stable form of metabolite in the tissues which have no glycogen or a small amount of it is a good working hypothesis.

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11. Anderson, R. J.: The Utilization of Inosite in the Dog, J. Biol. Chem. **25**: 391, 1916.

12. (*a*) Oppenheimer, S.: Ueber die Milchsäurebildung in der künstlich durchströmten Leber, Biochem. Ztschr. **45**:30, 1912. (*b*) Griesbach, W., and Oppenheimer, S.: Ueber Milchsäurebildung im Blut, ibid. **55**:328, 1913. (*c*) Embden, G., and Griesbach, W.: Ueber Milchsäure und Zuckerbildung in der isolierten Leber: I. Ueber den Abbau der d-Sorbose; II. Ueber das Schicksal des d-Sorbits und einiger anderer Hexite, Ztschr. f. physiol. Chem. **91**:284, 1914.

13. (*a*) Das, N., and Guha, B. C.: The Biological Oxidation of Inositol, Current Sc. **3**:157, 1934; abstracted, Chem. Abstr. **29**:1110, 1935; (*b*) Die Unwandlung von Inosit durch Rattengewebe, Ztschr. f. physiol. Chem. **231**:157, 1935. (*c*) Young, L.: Inositol and the Respiration of the Brain, Proc. Soc. Exper. Biol. & Med. **35**:507, 1936.

14. Starkenstein, E.: (*a*) Die Beziehungen der Cyklosen zum tierischen Organismus, Ztschr. f. physiol. Chem. **58**:162, 1908; (*b*) Die biologische Bedeutung der Inosit Phosphorsäure, Biochem. Ztschr. **30**:56, 1911.

Clinical Notes

A SPECIFIC TREATMENT FOR HERPES ZOSTER

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Herpes zoster of the eyes and forehead and sometimes of the side of the nose is one of the most stubborn diseases with which the ophthalmologist has to deal. The pain is often excruciating and sometimes cannot be relieved except by the administration of large doses of morphine.

For many years it has been known that when herpes occurred along the distribution of the fifth cranial nerve the eye of the side affected became involved. The sensory nerve fibers that supply the iris and some other parts of the eyeball are branches of the nasociliary nerve, which also supply the cornea; sensory branches of the fifth nerve also supply the eyelids, the conjunctiva, the caruncle and the lacrimal sac. Since herpes zoster follows the course of the fifth nerve and its numerous branches, one can see how the vesicles can be so widely distributed on the side of the face.

Herpes zoster is comparatively rare and may be mistaken for erysipelas. Such a mistake should not be made, especially by one who has ever seen a case of herpes. The symptoms are different. Herpes zoster usually develops to its entire extent at once and is seldom accompanied by fever, while erysipelas usually starts from one point and spreads and is accompanied by fever, which may reach from 103 to 106 F. Herpes involves only one side of the face, while erysipelas may spread over the entire face, the neck and the head. The onset of herpes is usually sudden, and the vesicles do not spread. Some patients suffer from pain for months after the eruption has subsided. After the vesicles have healed, there is a permanent scar. Sometimes paralysis of some of the ocular muscles occurs.

It has been our experience that we do not see patients with herpes zoster at the onset of the disease unless there is involvement of the cornea or the lids. Most patients are treated by their family physician and consult an ophthalmologist only after the condition is of one or two months' duration. However, in the past few months we have seen 2 patients with herpes in the acute initial stage.

ETIOLOGY

As to the cause of herpes zoster, we are still ignorant. We do not know whether it is of central or of peripheral origin. Some authors have suggested that it is caused by cold and some that it is caused by internal toxins. Stern [1] stated that it is caused by an infection passing from the sensory ganglion down the course of the sensory nerve to the

1. Stern, E. S.: Brit. J. Dermat. **49**:263, 1937.

skin. According to him, herpes zoster and chickenpox are the result of the same infection. He scarcely mentioned treatment. The condition is said to have been known to ophthalmic surgeons since 1866. A good description may be found in any textbook on ophthalmology, but most all of them deal briefly with the treatment.

TREATMENT

We recently reviewed the literature on herpes zoster and found that most writers consider medication to be unsatisfactory. Many report what they deem to be good results with the use of a solution of posterior pituitary. The library package of the American College of Surgeons gives little on the treatment. We have just reviewed the literature on the treatment of herpes zoster as sent us in the package library of the American Medical Association and find the following methods of treatment outlined.

Barksdale [2] reported good results with autohemotherapy. Vorhaus [3] used vitamin B_1 without positive results. Somers and Pouppirt [4] obtained good results with a solution of pituitary. Ruggles [5] in 1931 reported what seem to us better results with sodium iodide than with any other type of treatment. Beeson [6] reported excellent results with autohemotherapy. Franchini [7] reported a case of herpes zoster of the epiglottis. His treatment consisted of drugs to relieve pain. Pituitary preparations are used by a great many physicians, but the results are usually slow. Niles [8] reported several cases in which a solution of posterior pituitary was used; 5 of the patients were well in eleven days, 1 was improved but the pain continued, 4 were no better, and the results for 6 were unknown. We have never seen recurrent herpes zoster. In Norris and Oliver's [9] "System of Diseases of the Eye" there are three lines about treatment. Gifford [10] in his book on ocular therapeutics gave only a short outline on treatment.

About twenty years ago we began a treatment which has been used by us to the exclusion of all others, and that is the injection of diphtheria antitoxin. We administer 5,000 units of the antitoxin, and if necessary we give another dose of the same amount two days later. Usually the pain ceases after the first dose, and the inflammation disappears rapidly. In only 2 cases have we given a third dose, and in 1 we do not believe

2. Barksdale, E. E.: Virginia M. Monthly **64**:378, 1937.

3. Vorhaus, M. G.: Am. J. Digest. Dis. & Nutrition **3**:915, 1937.

4. Somers, M. R., and Pouppirt, P. S.: California & West. Med. **42**:370, 1935.

5. Ruggles, E. W.: Apparent Specific Effects of Sodium Iodide in Herpes Zoster, Arch. Dermat. & Syph. **23**:472 (March) 1931.

6. Beeson, B. B.: Autohemotherapy in the Treatment of Herpes Zoster, Arch. Dermat. & Syph. **18**:573 (Oct.) 1928.

7. Franchini, Y.: Semana méd. **1**:1323, 1937; Rev. Asoc. méd. argent. **51**:53, 1937.

8. Niles, H. D.: New York State J. Med. **32**:773, 1932.

9. Norris, W. F., and Oliver, C. A.: System of Diseases of the Eye, Philadelphia, J. B. Lippincott Company, 1897-1898.

10. Gifford, S. R.: A Handbook of Ocular Therapeutics, ed. 2, Philadelphia, Lea & Febiger, 1937.

that it would have been necessary. Some of the patients had had the pain for months. In a recent case pain had been present for nine months in spite of other treatment, but relief was obtained after the first dose of 5,000 units of diphtheria antitoxin. Other physicians whom we have induced to use the treatment report the same results. Some have asked if it was not the horse serum that was responsible for the cure. Since the use of concentrated diphtheria antitoxin, the results have been just as good.

While this paper was in preparation, we saw a patient with the most severe herpes zoster that it has been our lot to see.

W. R., a white man of about 50 years of age, was seen on Jan. 15, 1938, with extensive herpes involving both eyelids on the left side and extending up over the brow and forehead to the crown of the head. The blisters were the largest we had ever seen. The man was suffering from intense pain. The attack was of three days' duration when we saw him. We immediately gave him 5,000 units of diphtheria antitoxin. We saw him again on January 17; the acute pain was gone, and there were no new blisters. We gave him 5,000 units of the antitoxin, and on January 18 we gave him 5,000 more units. At that time the pain had ceased, and the blisters had begun to dry up. From then on he had no more pain and no increase of the herpes. We did not administer any more antitoxin, and the patient made a rapid recovery.

CONCLUSIONS

In our experience diphtheria antitoxin, as used by us, has proved to be a specific treatment for herpes zoster in the acute as well as in the chronic stage. It usually gives relief from pain within twenty-four hours, and the cure is permanent.

533 T. W. Patterson Building.

Ophthalmologic Review

DETACHMENT OF THE RETINA

SUMMARY OF THE MODERN OPINIONS

THOMAS D. ALLEN, M.D.
CHICAGO

To comprehend satisfactorily the status of the new and modern theories concerning detachment of the retina, one must have a reasonable understanding of the growth of the knowledge of this subject. To this end a few facts and opinions have been gleaned from encyclopedias and numerous reviews of the subject as well as from many original articles.

The early recognition of this condition, before the ophthalmoscope was invented, will be considered first. According to numerous authors, de Saint-Yves [1] in 1722 was the first to describe anatomically a condition called amotio sive sublatio retinae. After the appearance of this description, numerous articles were published in which the condition was mentioned, but no real advance was made for nearly a century. Ware [2] in 1805, Wardrop [3] in 1818 and Panizza [4] in 1821 are credited with histologic descriptions. These students examined specimens and differentiated hydrops subchoroidealis from hydrops subscleroticalis.

In 1830 Mackenzie [5] wrote of a watery fluid being present between the sclerotica and the choroid in cases of sclerotic staphyloma. He stated:

> There are also good grounds for believing that a similar effusion forms occasionally between the choroid and the retina. If the fluid . . . is not evacuated by puncturing the staphyloma, it may accumulate to such a degree as to press the retina before it, and having at last produced, by means of its continued pressure, an absorption of the vitreous humor, it will gather the retina into a cord. . . .

The condition was treated by thrusting a broad cataract needle toward the center of the vitreous, but not deeper than ⅛ inch (0.32 cm.).

This review is based on a course given at the American Academy of Ophthalmology and Otolaryngology.

1. de Saint-Yves, C.: Nouveau traité des maladies des yeux, Paris, P. A. Le Mercier, 1722, pp. 8 and 331.

2. Ware, J.: Chirurgical Observations Relative to the Eye, ed. 2, London, J. Mowman, 1805, vol. 1, p. 168.

3. Wardrop, J.: Essays on the Morbid Anatomy of the Human Eye, ed. 2, London, A. Constable & Co., 1818.

4. Panizza, B.: Annotazioni anatomico-chirurgiche sul fungo midollare dell'occhio, Pavia, P. Bizzoni, 1821.

5. Mackenzie, W.: A Practical Treatise on the Diseases of the Eye, London, Longman [and others], 1830, p. 460.

The first person seemingly to recognize retinal detachment in situ was von Chelius,[6] who in 1839 observed it through a dilated pupil as a white membrane.

After the invention of the ophthalmoscope, Coccius, van Trigt, Arlt[7] (1853) and von Graefe[8] (1854) described the ophthalmoscopic appearances of detachment of the retina.

It is interesting to note that in the first article by von Graefe on this subject, which appeared in the first volume of the *Archiv für Ophthalmologie,* trauma is noted as the cause of retinal detachment in certain cases; also chorioretinitis and a tendency to bleeding (hemorrhoids, epistaxis and other conditions in which bleeding is often severe). This article contains one of the first descriptions of retinal detachment after the invention of the opthhalmoscope. Von Graefe noted the color of the detached areas as well as the position of the detachment. Some of the areas were yellow, some white and some red. He expressed the belief that some detachments must be due to hemorrhage, the blood subsequently absorbing, at least in part. In this he differed with Arlt, who had contended that a choroidal effusion was the causative force.

Stellwag[9] disproved this hemorrhagic theory by his anatomic investigations.

In 1857 Müller[10] called attention to the existence of contracting connective tissue in the vitreous, which he considered an important etiologic factor. In the same year von Graefe[11] drew attention to the association of retinal detachment with myopia. He expressed the belief that the outer tunics of the eye were more distensible than the retina, which was therefore pulled away as the eyeball enlarged with the development of myopia.

In 1869 Knapp[12] described a hole in the macula, and the following year de Wecker[13] emphasized the frequent occurrence of tears in cases of spontaneous detachment of the retina.

"Leber noticed tears and detachment occurring together in eyes with retained foreign bodies. He introduced, aseptically, metallic bodies into the vitreous and watched the formation of retinal tears and after a few

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6. von Chelius, M. J.: Handbuch der Augenheilkunde, Stuttgart, E. Schweizerbart, 1839, vol. 2, p. 366.

7. Arlt, F.: Die Krankheiten des Auges, Prague, F. A. Credner & Kleinbub, 1855, vol. 2, p. 158.

8. Von Graefe, A.: Arch. f. Ophth. (pt. 1) **1**:362, 1854.

9. Stellwag von Carion, C.: Die Ophthalmologie, vom naturwissenschaftlichen Standpunkte aus bearbeitet, Erlangen, Ferdinand Enke, 1856, vol. 2, p. 100.

10. Müller, H.: Ztschr. f. wissensch. Zool. **8**:1, 1857.

11. von Graefe, A.: Arch. f. Ophth. (pt. 2) **2**:277, 1857.

12. Knapp, J. H.: Arch. f. Augenh. **1**:1, 1869.

13. de Wecker, L.: Traité des maladies du fond de l'oeil, Paris, A. Delahaye, 1870, p. 153.

days, extensive detachments of the retina."[14] Lindner[15] recently remarked: "He [Leber] was the first to say that vitreous detachment precedes detachment of the retina and is caused by peripheral choroiditis." Leber[15a] divided retinal detachments into three categories: (*a*) acute, characterized by shrinkage of the vitreous and an outpouring of fluid through retinal tears produced by traction of strands of vitreous; (*b*) chronic, characterized by shrinkage of the vitreous without tears and without fluid passing through the retina, and (*c*) a type characterized by the formation of a cyclitic membrane with shrinkage of the vitreous.

From time to time increments were added to the knowledge of the subject, but it was not until 1887 that Nordenson[16] made a fairly satisfactory grouping of cases; consideration of these groups led him to develop an interesting theory. He stated that examination of 119 persons with separation of the retina revealed one rent or more in over one third of the group. According to his theory, the primary lesion is located in the uveal tract; the uveitis is followed by fibrillary metamorphosis and subsequent shrinkage of the vitreous. The vitreous becomes firmly adherent to the retina and in contracting not only detaches the retina from its choroidal bed but tears it; through these openings, often minute, the posthyaloid effusion passes. If the detachment occurs in the upper part of the globe, the fluid gravitates downward and further tends to increase the detachment.

Leber's first theory[14] was based on previously established facts; among these were Arlt's finding of fluid back of the vitreous in cases of advanced myopia and Elschnig's qualification that this occurred in less than half of these cases. (Therefore, detachment is not a rule in cases of high myopia.) According to Leber's first theory, myopia plus degeneration of the vitreous and contraction of the degenerated fibrils of the vitreous which are fastened to the retina and to the pars plana of the ciliary body cause tearing of the retina; fluid passes through the tear and elevates the retina. The degenerative changes develop as follows: Far forward on the ciliary body small inflammatory lesions are accompanied by a proliferation of epithelial cells which migrate onto the fibrils of the vitreous; here they have too little nourishment to maintain life, so degenerate and shrink—"fibrillary degeneration of the vitreous." When sufficient shrinkage occurs, the fibrils contract and

14. Leber, T.: Tr. Internat. M. Cong. (1881) **3**:15, 1882; quoted by Anderson, J. R.: Detachment of the Retina, New York, The Macmillan Company, 1931, p. 64.

15. Lindner, K.: Lectures before the Chicago Ophthalmology Society, 1935.

15a. Leber, cited in Wood, C. A.: American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1919, vol. 15, p. 11212.

16. Nordenson, E.: Die Netzhautablösung: Untersuchungen über deren pathologische Anatomie und Pathogenese, Wiesbaden, J. F. Bergmann, 1887, p. 4.

tug on the retina; this tugging produces the predetachment photopsia. Then when a fluid exists behind the vitreous some jerky movement of the eye causes these fibrils to tear the retina.

Leber's second theory, that of "preretinitis" (which theory was evolved in association with von Hippel[17] in 1900 and 1908), was developed to explain the presence of small inflammatory masses, occasionally seen, uniting the vitreous and the internal limiting membrane. According to this theory, the shrinkage of the preretinal exudate wrinkles the retina, and fluid then gathers beneath it, either through a retinal tear or ex vacuo by transudation.

It is often difficult, it has seemed to me in this study, to determine exactly how one theory differs from another; this is due in part to the point of view of the author considered and to his understanding of the point of view of previous authors, and in part to the fact that the later point of view is different from that of the earlier authors whose works are reviewed here. Also, many reviewers inaccurately attribute to one man a theory propounded by another author. Thus, for example, Siergrist, of Berne, Switzerland, in his discussion of a paper by Gonin which appeared in 1919, gave Gonin credit for the idea of primary chorioretinitic foci in the periphery of the fundus leading to the formation of adhesions of the vitreous, with subsequent shrinkage and the development of retinal folds and tears. These ideas were part and parcel of several previous theories.

Having established this foundation, I shall next consider Gonin's[18] theories, although in so doing some forward steps are necessarily being omitted. Gonin, however, by startling the world in 1919 and the next few years, focused attention on his contribution, which sums up and gives point to many of the previous findings.

Gonin tried to differentiate the various kinds of detachment and the factors producing them and called attention to the usual lack of understanding of these several factors. A careful history of the patient and a detailed examination of retinal detachments at consecutive stages led to a better understanding of the pathologic process, and to the proper treatment. Gonin divided detachments into the following four large groups on the basis of the etiologic factors: (1) those due to pulsion (*soulévement*), (2) those due to traction, (3) those due to distention and (4) those due to depression (diminution of the size of the globe). According to him, treatment is seldom successful for the last two types of detachment; for the first type, the treatment is mainly directed to the cause. Most of this paper was devoted to a discussion of this type of detachment. In other papers, Gonin reviewed many previous state-

17. von Hippel, E.: Arch. f. Ophth. **51**:132, 1900; **68**:38, 1908.
18. Gonin, J.: Ann. d'ocul. **156**:281, 1919.

ments, agreeing with some and disagreeing with others. His principal contribution to the subject was that the reapplication of the retina is permanent only when the traction exerted on the retina by the vitreous has ceased or is counterbalanced by a sufficient adherence of the retina to the choroid. According to him, such a reapplication can be conceived of only after the closure of the one or more tears of the retina. Such closure was the aim of the treatment, especially closure brought about by the production of an extensive chorioretinal adhesion involving all of the edges of the tears. Gonin's classic method of treatment is well known. He himself has modified his theory from time to time in more or less important details; other investigators have added their quota also; many have overemphasized the importance of these modifications.

I shall now touch on some of these modifications that seem to be of value and shall especially consider the various views on the production of the hole, it having been agreed that a hole is a necessary prerequisite for detachment. In Arruga's [19] recent work on "Detachment of the retina" (published in Spanish and in English), three theories of this mechanism are illustrated, Gonin's, Lindner's and Arruga's. The differentiation appears in parallel columns:

Gonin	Lindner	Arruga
19. Retraction of the vitreous with its separation from the retina mainly superiorly.	23. Liquefaction of the vitreous in the posterior part.	27. Adherence of the vitreous to the retina. It may exist for a long time before energetic trepidation of the vitreous or the motions of the eyeball produce the retinal rent.
20. Retraction of the vitreous with a vitreoretinal adhesion superiorly.	24. Detachment of the vitreous with adhesion above.	28. Ocular movements compel the vitreous to strike the edges of the hole, thus forcing the vitreous behind the retina.
21. Formation of a retinal rent due to increased traction of the vitreous.	25. Formation of a tear with a flap.	29. An elevation of the retina caused by choroidal exudate. This elevation is localized by a chorioretinal adherence.
22. Displacement of subretinal fluid downwards.	26. Formation of a retinal hole with floating operculum.	30. A rupture of the retina at the point where adhesion between the choroid and retina was present. Ocular movements facilitate the progress of the detachment.

19. Arruga, H.: Detachment of the Retina, translated by R. Castroviejo, New York, B. Westerman, 1936, pp. 26-27.

In brief, Gonin stated that the shrinking vitreous causes no trouble unless it is fastened securely to the retina (not to the choroid) in a favorable spot, so that in shrinking it will pull the retina loose and tear it at that spot, allowing the retrovitreous fluid to pass through the tear. Lindner emphasized the frequency with which slit lamp examination reveals an optically empty space behind the vitreous, especially in cases of myopia. According to him, if the shrinking vitreous is adherent to the retina, it may pull a flap loose, causing a horseshoe tear, or it may pull off a small button of retina, causing a hole. Arruga expressed an inclination to minimize the effects of shrinking vitreous and emphasized the effect of sudden motions of the eyeball on a diseased retina which is in one or more places slightly adherent to the vitreous. He stated:

> As a result of previous inflammation (chorioretinitis) [20] or degenerative processes (atrophy and cystoid degeneration), the retina becomes adherent to the vitreous in certain places. At the same time it becomes thinner, weaker and retractile, a condition found in all tissues following inflammation or degenerative atrophy. Then, a blow on the head or the eye, even movements of the eyeball may make the adhesion of the vitreous produce a rent in the retina.

It is difficult to decide who really was the first to emphasize the effect of motion; certainly, almost simultaneously the use of pinhole disks in treatment was adopted by nearly all clinics. Arruga further stated that several favoring conditions have to be present for a retina to become detached: (1) hole, (2) retractibility of the retina, (3) adhesion of the retina and the vitreous and (4) an inciting cause, such as a blow on the head or a sudden movement of the eyeball. He emphasized the fact that holes are often present without detachments resulting; e. g., after equatorial sclerotomy (therapeutic) the retina does not detach itself. Detachment associated with pregnancy and nephritis ordinarily occurs without the formation of holes, and spontaneous healing results.

Anderson,[21] in summing up many opinions, brought out the relation between the degenerative changes in the retina, particularly the anterior third, in cases of myopia and those in cases of retinal detachment. In both conditions the retina is thin and atrophic, especially in the region of the ora serrata, where the formation of a hole is a frequent finding. Also the vitreous is abnormal, degenerate. Anderson was undoubtedly influenced in this opinion by the accumulated experience of many leaders who from the time of Blessig, in 1855, to that of Kopmczinski, in

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20. A footnote in Arruga's article at this point reads as follows: "If, experimentally, an inflammation of the membranes of the eye is produced by irritating the sclera, not only do the membranes become adherent to each other, but the vitreous body also becomes adherent at the point where there was inflammation."

21. Anderson, J. R.: Detachment of the Retina, New York, The Macmillan Company, 1931, p. 39.

1929, reported on cystoid degeneration of the retina. Hanssen [22] and others collected considerable data on the prevalence of cystoid degeneration of the retina and reasoned that when these cysts broke holes would form. Hanssen expressed the belief that the cysts would break easily if the internal limiting membrane covering the retina were adherent to the vitreous and a slight injury were received at that spot. In this he agreed with Gonin's statement of four years before.[23]

In 1929 Pressburger [24] reported on an extensive study of the cystoid changes in the retina. He stated that they could occur at any age but were most frequent in senile and myopic eyes, and that they were seldom found farther than 7 mm. from the ora serrata. According to him, vitreous pulling on such degenerated tissue would cause tears and holes, especially if the cyst were unusually large.

The part played by trauma has been stressed by some authors. Gifford [25] expressed the belief that detachment may occur many years after an injury. Others have stated that there are few patients who cannot, if pressed, recall some injury; for example, a blow on the head or eye, rubbing of the eyes, sudden motion of the eyes, such as one makes when an unexpected noise causes a person to look around suddenly, or a fall or a coughing spell. Recently Walker [26] pointed out the effect of a sudden movement of the eyes up and to the right on the upper outer quadrant of the left retina, as caused by the combined action of the left internal rectus muscle and the inferior oblique muscle acting against the superior oblique muscle. He expressed the opinion that a sudden jerk of the eyes as in fright would easily whip a retina loose if certain favorable factors were present. Yet if these favorable factors are not present, a detachment does not result, as, for example, in cases of old choroidal tears and in most cases of cataract extraction.

And finally I shall add a few words about the hole in the macula and its relation to detachment. As previously stated, Knapp [12] in 1869 was the first to describe a hole in the macula, but its significance was not appreciated even by Middleton,[27] who in 1919 reported detachment of the retina and a macular hole in a soldier who gave a history of trauma. Vogt [28] in 1924 reported a case of retinal detachment and iritis in a man in whom the retinal tear became suddenly larger and

22. Hanssen, R.: Klin. Monatsbl. f. Augenh. **74**:778, 1925; **75**:344, 1925.

23. Gonin, J.: Klin. Monatsbl. f. Augenh. **67**:316, 1921.

24. Pressburger, E.: Ztschr. f. Augenh. **68**:331, 1929.

25. Gifford: Personal communication to the author.

26. Walker, C. B., in discussion on Spiegel, E. A., and Scala, N. P.: Ocular Disturbances Associated with Experimental Lesions of the Mesencephalic Central Gray Matter, Arch. Ophth. **18**:614 (Oct.) 1937.

27. Middleton, A. B.: Am. J. Ophth. **2**:779, 1919.

28. Vogt, A.: Klin. Monatsbl. f. Augenh. **72**:335, 1924.

was associated with acute hypotony. The following year [29] he established the clinical entity "cystoid macular degeneration," or honeycomb macula. This is similar to Blessig's cystic degeneration of the peripheral portion of the retina. This condition is not easily seen in the macula without the aid of the red-free light. These holes are assuming more importance because of Lindner's recently developed "undermining" method of chemical cautery.

The macular holes may be primary, the result of contusion either directly, such as is caused by a blow from a stone or fist or from a bullet or stick, or indirectly, with an interval during which inflammatory or degenerative processes develop, or they may be secondary to the detachment itself, being caused by toxemia of the interretinal fluids or by wavy motions of the detached retina as the eye moves. Since the macula is the most delicate part of the retina, the retroretinal fluid washing against it would cause a tear in this portion first. Naturally, such a secondary hole must be treated just as any secondary or primary hole must be treated, that is, by the production of seclusive chorioretinitis.

29. Vogt, A.: München. med. Wchnschr. **73**:1101, 1925.

Correspondence

A NEW METHOD FOR REBUILDING A LOWER LID

To the Editor:—An article by Dr. Wendell L. Hughes entitled "A New Method for Rebuilding a Lower Lid" in the June 1937 issue of the ARCHIVES (page 1008) has just been called to my attention by one of my associates.

I am surprised that this article has escaped my notice for so long, but it seems impossible to see all of the contributions on reconstructive surgery appearing in the various journals.

Dr. Hughes offered this technic as an original contribution and, undoubtedly, has received much credit for an ideal method on this basis. It is, however, a faithful description of the method presented before the Société française d'ophtalmologie by Dr. L. Dupuy-Dutemps, ophthalmologist at Hôpital Saint-Louis, Paris, in May, 1927.

The method is described in detail, with full illustration of the various steps of the procedure and a photograph of a completed operation, on pages 161 and 162 of my text "Reconstructive Surgery of the Head and Neck," published by Thomas Nelson & Sons, New York, in 1928.

Dr. Hughes' only variation of this method is the replacement of the skin resulting from the excision of the lid. He elevates the facial skin beneath the eye to cover the new lower lid instead of utilizing normal skin from the upper lid. He adds grafted eyelashes after the method described in detail by John Wheeler in a paper entitled "Plastic Operations About the Eye; Suggestions for Several Important Conditions" in the 1922 *Transactions of the International Congress of Ophthalmology* (page 356), but fails to mention Dr. Wheeler.

FERRIS SMITH, M.D., Grand Rapids, Mich.

To the Editor:—Dr. Smith's letter has come at a time when I am in the midst of conducting a more thorough search of the literature. When this is completed, it will be offered for publication.

There are several methods for reconstruction of the lid in which the structures of the lid are used, one of the earliest being that of Landolt, followed by that of Kollner and of Dupuy-Dutemps. The method reported in my paper differs from each one previously described.

I wish to thank Dr. Smith for calling my attention to Dr. Dupuy-Dutemps' procedure, which was not mentioned in the list of references supplied me from the Library of the Surgeon General's Office at Washington, D. C.

WENDELL L. HUGHES, M.D., Hempstead, L. I.

SIMPLIFICATION OF THE O'CONNOR CINCH OPERATION

To the Editor:—The article by Dr. M. E. Smukler on simplification (?) of my cinch operation in the June issue of the ARCHIVES (page 930) is most pleasing to me because it indicates that after twenty-seven years interest in it has reached the Atlantic Coast and because in the

discussion Dr. L. F. Appleman emphasized some of its advantages, especially the *absence of reaction.* Most eastern physicians in mentioning the operation emphasize just the opposite idea.

In devising his instrument to facilitate (?) the passing of the dermal shortener, Dr. Smukler has fallen into the common error that I always divide the tendon into four strips. In describing my operation I have shown the tendon split into four strands merely for diagrammatic purposes. I often split it into five, six or even up to ten or twelve strips, depending on the result desired. Hence a four-pronged separator really does not simplify the operation. Moreover, when Dr. Smukler learns the knack of passing the shorteners, I think that he will admit that the time spent in placing his separator is little less than the time spent in passing the shorteners. This knack consists merely in pulling aside the strip that has been looped first, then picking up the exposed second strip and so on across the entire width of the tendon.

As to length of metallic X coating, I like the ½ inch (1.3 cm.) length for the same reason that one does not use 1½ inch (3.8 cm.) needles for conjunctival or corneal sutures. The shorter coating, when bent ¼ inch (0.6 cm.) from its end, goes between the strips of tendon with less discomfort to the patient.

In general, the fewer special instruments needed to do a particular operation the better. The flat hook is really the only special instrument needed in the cinch operation, and it has come into such universal use in all types of operations on muscles that at the present date it can hardly be called a special instrument.

RODERIC O'CONNOR, M.D., San Francisco.

News and Notes

UNIVERSITY NEWS

Postgraduate Courses, Harvard University Medical School.—Postgraduate courses in ophthalmology at the Harvard University Medical School are divided as follows: (1) courses open to general practitioners, dealing with the use of the ophthalmoscope and ocular complications in general disease; (2) a course in the fundamental sciences in ophthalmology, open to beginners in the specialty and devoted to a study of the essentials of embryology, anatomy, pathology, physiologic optics, physiology, chemistry and pharmacology; (3) courses in clinical ophthalmology, ocular pathology and physiologic optics, with a four weeks' intensive course on recent advances in ophthalmology, open to advanced students.

Obituaries

JAMES A. SPALDING, M.D.

1846—1938

Dr. James A. Spalding was born at Portsmouth, N. H., Aug. 20, 1846, the son of Lyman Dyer and Susan Parker Spalding, and died at his home in Portland, Me., on Feb. 27, 1938, in his ninety-second year. He was a grandson of Dr. Lyman Spalding, the founder of the Pharmacopeia of the United States, which was responsible for effecting uniform writing of prescriptions. After graduating from Dartmouth College, he received the degree of Doctor of Medicine from Harvard in 1870. He had a defect in hearing, which became gradually worse, and he was advised to specialize in diseases of the eyes and of the ears. After he had studied these specialties abroad, he started to practice in Portland in 1873. He became ophthalmologist and otologist to the Maine General Hospital and took an active part in the development of the practice of these two specialties and helped to found the ophthalmologic and otologic clinics at Augusta, Bangor and Portland.

He was a member of the American Ophthalmological Society, the American Academy of Medicine, the American Academy of Ophthalmology and Otolaryngology, the Maine Medical Association, of which he was once president, and the Cumberland County Medical Association.

Dr. Spalding was the author of many medical articles and also wrote several books, including "Maine Physicians in 1820." He married Miss Sarah Chase Shepley, of Boston, in 1882.

In addition to his interest in the two specialties which he practiced, he was a linguist, being unusually proficient in German, Italian and French and also having studied Spanish and Gaelic. He had an analytic mind, and few things escaped his keen sense of humor.

He early came under the influence of Dr. Herman Knapp and through many years was of the greatest help in the publication of the ARCHIVES OF OPHTHALMOLOGY and the *Archives of Otology*.

Notwithstanding his loss of hearing, his career has been a most distinguished one. On the occasion of a dinner in his honor, on Sept. 19, 1931, Dr. Edwin W. Gehring stated that Dr. Spalding was the "Nestor of the medical profession of Maine; a man of broad culture, profound learning, and wide human sympathies, whose perennial youth startles and delights us."

ARNOLD KNAPP.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

HISTOLOGY OF BOWMAN'S MEMBRANE IN CASES OF GLAUCOMA. G. TALBOT, Brit. J. Ophth. **22:** 210 (April) 1938.

In 1937 Wolff and Lyle reported finding hemispherical bodies in Bowman's membrane in an eye excised for chronic glaucoma. In 14 cases, in all of which there were other evidences of glaucoma, Talbot found these bodies in Bowman's membrane. In 4 cases in which there were evidences of glaucoma the bodies were not found, nor were they observed in any case in which tension was presumed to be normal.

The bodies are best brought out by Verhoeff's stain or by phosphotungstic acid and hematoxylin.

Their appearance is constant. They are small structureless hemispheres, measuring about 10 microns, at the base, which is attached to the corneal epithelium. Sometimes one gets the impression that they exceed a hemisphere and that at least two thirds of a globe can be seen.

The presence of such bodies is indicative that the tension is still elevated and is of at least three days' standing.

Wolff suggested that the bodies might be of a hyaline nature in connection with the nerves penetrating Bowman's membrane. The author wonders if they are lacuna-like spaces around the nerve fibers.

One illustration accompanies the article.

W. ZENTMAYER.

Biochemistry

SEROLOGIC RESEARCH: SPECIFIC PROPERTIES OF THE LENS. E. WOLLMAN, P. GONZALES and P. DUCREST, Compt. rend. Soc. de biol. **127:** 668, 1938.

Confirming the work of Uhlenhuth, the authors found that a mammalian lens antiserum though inactive against a lens suspension of the same species precipitates in dilute suspension lenses from all other mammals and birds but affects a suspension of fish lens slightly, if at all. However, if the lens antiserum is derived from fish, the result is positive against all vertebrate lenses, though in varying degree—a dilution of 1:80,000 for the lenses of ray, 1:40,000 for the lenses of coalfish and 1:10,000 for the lenses of mammals and birds. On the other hand, the lens of the cephalopod does not react at all with vertebrate lens antiserums, or vice versa. The chemical constitution of the lens is hence probably somewhat similar throughout the vertebrate kingdom.

J. E. LEBENSOHN.

Conjunctiva

LYMPHOID REACTION OF THE BULBAR CONJUNCTIVA: REACTION TO TUBERCULIN. J. L. PAVÍA, Rev. oto-neuro-oftal. **12:** 36 (Feb.) 1937.

After reference to 4 similar cases previously reported by him, Pavía presents 4 new cases of a peculiar vitreous-like formation in the bulbar

conjunctiva of patients with active tuberculous lesions and a susceptibility to tuberculin injected intradermally.

The process is characterized mostly by a slight photophobia and lacrimation, a superficial vascular injection at the site of the lesion, dilatation of the lymphatics accompanying the blood vessels, a slight localized chemosis, semitransparent formations in the vitreous (from 0.3 to 3 mm. in diameter), which are easily overlooked, and rarely some circumcorneal injection.

Histologic examination in 1 case showed a diffuse lymphoid infiltration, and biopsy of an accompanying follicular infiltration of the lid showed a scattered similar slight follicular formation.

In all except 1 case the intradermal injection of tuberculin was followed by a marked local reaction and a prompt disappearance of the ocular lesion. In the case in which there was a negative Mantoux reaction after three trials, the ocular lesion promptly disappeared after the injection of tuberculin. In this case there was a preexisting elevation of temperature and a persistent cough.

C. E. FINLAY.

Cornea and Sclera

A PRELIMINARY REPORT OF A CASE OF KERATOCONUS, SUCCESSFULLY TREATED WITH ORGANOTHERAPY, RADIUM, AND SHORT-WAVE DIATHERMY. H. L. HILGARTNER, H. L. HILGARTNER JR. and J. T. GILBERT, Am. J. Ophth. **20**: 1032, 1937.

A case of keratoconus associated with hypothyroidism is reported in an 18 year old girl. Short wave diathermy, the use of drops of ethylmorphine hydrochloride and mercurochrome at home, radium treatment and thyroid therapy resulted in improvement of the vision in the right eye from hand movements at 1 foot (30 cm.) on Jan. 2, 1937, to 20/100 on February 19. The authors consider the diathermy responsible for the improvement.

W. S. REESE.

TREATMENT OF MOOREN'S ULCER WITH LIVER EXTRACT. C. J. CANTILLON, Brit. M. J. **1**: 71 (Jan. 8) 1938.

Two cases of Mooren's ulcer are reported in which an injection of liver extract was given daily and then every other day for three weeks. In addition, Metri's cautery (from 80 to 85 F.) was applied to the margins of the ulcer for one minute every second day.

In the second case the treatment was interrupted, and a relapse occurred. Treatment was again begun and continued with fortnightly injections.

ARNOLD KNAPP.

SENILE DEGENERATION OF THE SCLERA AT THE INSERTION OF THE OCULAR MUSCLES: REPORT OF A CASE. H. GASTEIGER, Klin. Monatsbl. f. Augenh. **98**: 767 (June) 1937.

Gasteiger refers to the publications of Pillat, Kiss and Kreibig on this subject and reports the case of a woman aged 78 with bilateral senile degeneration of the sclera at the insertion of the extrinsic ocular muscles. Dark, glassy looking bands, running fairly perpendicularly,

were observed in the right eye near the insertion of the horizontal and the inferior rectus muscle. These bands had a straight outline toward the cornea, but they were slightly humpy toward the temples. Slit lamp examination showed the unevenness to be produced by tendons of the muscles. A similar condition existed in the left eye, but for the absence of the band near the inferior rectus muscle. Scleral transillumination showed a weak red reflex over the temporal band of each eye, whereas the other bands remained dark. This picture differs from that reported by Pillat, who observed illumination of all bands, the appearance being that of bright red windows. Experiments on pigs' eyes dehydrated with glycerin showed that this change was gradual but not essential. Gasteiger concludes that conditions of dehydration are responsible for these phenomena in human eyes also.

K. L. STOLL.

Experimental Pathology

EXPERIMENTAL DINITROPHENOL CATARACT IN ANIMALS. H. SOHR, Arch. f. Ophth. **138:** 332 (Feb.) 1938.

The chief American literature on dinitrophenol cataract is reviewed. The author fed dinitrophenol to guinea pigs for two and a half months, giving from 30 to 40 mg. per kilogram of body weight by stomach tube two or three times a week. Of the 25 guinea pigs treated in this way, 17 died during the feeding period. The remaining 8 animals were observed for four months, during which time 5 more died. The remaining 3 animals retained macroscopically and microscopically normal lenses.

P. C. KRONFELD.

THE PERMEABILITY OF THE CAPSULE OF THE LENS AND THE DEXTROSE CONTENT OF BLOOD, AQUEOUS AND LENS IN ANIMALS WITH GALACTOSE CATARACT. T. SASAKI, Arch. f. Ophth. **138:** 365 (Feb.) 1938.

Certain phases of the carbohydrate metabolism of rats fed galactose and suffering from galactose cataract were studied. The reducing sugars of the whole blood, of the aqueous and of the lens were determined by the method of Hagedorn and Jensen, and the results were expressed in terms of dextrose. The cataractous lenses were incubated in saline solution for five hours, and the amounts of dextrose which were used up by the lenses during this period (glycolysis) and the amounts which diffused out of the lenses into the saline solution were determined. The dextrose content of the whole blood and that of the aqueous were found to be considerably higher for the rats fed galactose than for the control animals. The cataractous lenses, however, contained about the same amount of simple sugars as the lenses of the control animals. From these data the author concludes that in the animals with galactose cataract the osmotic pressure of the aqueous was higher than that of the lens, which difference might play a part in the production of the cataract. Incubated in saline solution, the cataractous lenses gave off more sugar than normal lenses, a phenomenon which indicates increased permeability of the capsule of a cataractous lens. If dextrose was fed young rats instead of the galactose of the cataract-

producing diet, no cataract developed; the dextrose contents of blood, aqueous and lens were found to be increased. No difference in osmotic pressure prevailed between aqueous and lens, and the permeability of the capsule of the lens remained normal, as far as its condition could be judged from the loss of sugar during incubation.

P. C. KRONFELD.

General

THE SIGNIFICANCE OF HEREDITY IN OPHTHALMOLOGY: PRELIMINARY SURVEY OF HEREDITARY EYE DISEASES IN TASMANIA. J. B. HAMILTON, Brit. J. Ophth. **22**: 19 (Jan.); 83 (Feb.); 129 (March) 1938.

After a brief historical sketch and some vital statistics on Tasmania, Hamilton tabulates the associated hereditary defects of the eye with hereditary defects in other parts of the body and also possible hereditary ocular defects.

These articles constitute a treatise on the significance of heredity in ophthalmology and do not lend themselves to abstracting.

W. ZENTMAYER.

USE OF CONTACT GLASSES FOR COSMETIC PURPOSE. S. K. MUKERJEE, Brit. J. Ophth. **22**: 43 (Jan.) 1938.

Unsuccessful attempts had been made to darken the cornea of a girl aged 16 in order to conceal leukomatous changes. When she was seen by the author the cornea was slightly staphylomatous, and the iris tissue was entangled in the wound. An excellent cosmetic effect was obtained with the use of a contact glass, which was painted on the inside to match the color of the other eye.

The article is illustrated. W. ZENTMAYER.

INFLUENCE OF LIGHT ON ARTERIAL TENSION. H. VIGNES, Compt. rend. Soc. de biol. **127**: 768, 1938.

Some obstetricians have been clinically impressed with the adverse influence of light in cases of eclampsia and have insisted on keeping their patients in a darkened room. Experimentally, sudden passage from darkness to a well lighted room (300 watts at 2 meters) induced an increase in the blood pressure of from 5 to 15 mm., especially in pregnant women, among whom those menaced by eclampsia were particularly sensitive.

J. E. LEBENSOHN.

General Diseases

DYSOSTOSIS MULTIPLEX WITH SPECIAL CONSIDERATION OF THE OCULAR CONDITION: REPORT OF A CASE. H. GASTEIGER and L. LIEBENAM, Klin. Monatsbl. f. Augenh. **99**: 433 (Oct.) 1937.

Dysostosis multiplex, a rare osteodysplasia, is a degenerative dysostosis of a hyperplastic type. One of 2 female twins aged 13 was normal; the family history was unimportant. The other twin was born in breech presentation, but developed normally up to the age of 2½ years, when

her size remained behind that of her sister. The growth in height stopped entirely when she was 7 years old. She had a massive head and was disproportionably dwarfed; a deformed thorax, a gibbus in the upper lumbar portion of the spine, a protruding abdomen, deformed extremities and a clumsy gait were noted. The function of the joints was limited, and the mouth could not be fully opened. The inner organs were normal. A detailed description of the roentgenograms is given. No changes of the skull were found aside from bony connections between the anterior and the middle clinoid process and between both anterior and posterior clinoid processes. There was an absence of ocular changes except for corneal nubeculae. Under the slit lamp these nubeculae appeared to be composed of numerous small, dot-shaped grayish-white opacities; they were evenly distributed in all layers of the cornea, but least so in Bowman's membrane. The transparency and sensitiveness of the cornea and the field of vision were normal; vision was 5/5 after correction of hyperopia of 5 and 6 D., respectively. No further pathologic changes were observed later. Pain in the back and dyspnea disappeared, while the function of the extremities increased under systematic active and passive exercises. Ample space is devoted to the differential diagnosis, and the etiologic factors are discussed. The authors conclude that dysostosis multiplex is a congenital disturbance which leads to disproportionate dwarfness. The causal genesis of the disease is unknown.

K. L. Stoll.

Glaucoma

The Association of Cyclodialysis with Fistulizing Operations in Certain Cases of Glaucoma. M. G. Besso, Ann. di ottal. e clin. ocul. **65:** 625 (Aug.) 1937.

On a number of eyes with secondary, subacute or chronic absolute glaucoma in which vision had been reduced to zero or nearly zero the author performed corneoscleral trephining according to the method of Elliot. However, on completion of the sclerotomy, he inserted the Elschnig spatula in the trephine opening and separated the ciliary body from the sclera in an area corresponding to that covered by the usual cyclodialysis. This was followed by basal iridectomy, after which the flap was sutured. The tension in eyes with the subacute and chronic forms of glaucoma was usually maintained below normal by this procedure, while in those with glaucoma secondary to recent uveitis, exacerbations of inflammation occurred, which prevented the desired result. Reasons are given which may lead to the adoption of the procedure for other types of glaucoma. Patients on whom this operation was performed have been followed for from six to twelve months.

S. R. Gifford.

Injuries

Superficial and Deep-Seated Disciform Keratitis in Caisson Workers After Exposure to Hydrogen Sulfide: Report of Cases. K. Hartmann, Klin. Monatsbl. f. Augenh. **99:** 456 (Oct.) 1937.

In the first chapter of this publication Hartmann refers to ocular lesions produced by hydrogen sulfide as a disease observed more frequently in recent years. It occurs in workers in factories producing

artificial silk, sugar, soap and glue and also in those employed in tanneries, dye works, mines, and sulfur baths. The injuries produced by this chemical have been recognized as an occupational disease through the effort of Thies. Previous publications in point are recorded.

Six cases of this condition in caisson workers are reported. The location in which the lesions occurred is described at the beginning of the second chapter, and a detailed clinical history of each case follows. The men were working at a depth of from 8 to 13 meters below the surface of the North Sea; the ocular disease occurred mostly when they were working in a layer composed of loam and shells, in which sulfurous gases abounded. Recovery followed when local treatment was administered and the workers were placed in well aired surroundings; the ocular symptoms recurred as soon as the men returned to the lower strata. The symptoms consisted chiefly of punctate defects and vesicular eruptions in the cornea, localized within the area of the palpebral fissure. The sensitiveness of the cornea was reduced. No complications of the intraocular organs developed. A resistance to the fumes of hydrogen sulfide could not be acquired.

The prognosis and course were favorable in all but 1 case, in which a deep-seated disciform keratitis left a dense scar. Hartmann discusses the possibility of an infection with the herpes virus in this case, because the lesions produced by hydrogen sulfite are usually limited to the corneal epithelium.

Suggestions are given for the prevention of this type of keratitis. Among them are: elimination of those workers who have previously suffered from diseases of the respiratory tract and corneoconjunctival diseases; proper ventilation and fresh air containing not more than 0.05 per cent of sulfurous gases; air-tight goggles, and shifts of not more than three or four hours.

K. L. STOLL.

Lens

SPHEROPHAKIA, LUXATION OF LENSES, AND SECONDARY GLAUCOMA RELIEVED BY EXTRACTION OF LENSES. I. JACOBS, Am. J. Ophth. **20:** 1042, 1937.

Jacobs discusses spherophakia and dislocation of the lens and the aggravation of glaucoma in such cases by the use of miotics. He cites the case of a 17 year old boy who had bilateral dislocation of the lens. Both lenses eventually became dislocated into the anterior chamber, and secondary glaucoma ensued, which was relieved by the administration of a few drops of a 1 per cent solution of atropine. Both lenses were successfully removed.

W. S. REESE.

PSYCHOSES COMPLICATING RECOVERY FROM EXTRACTION OF CATARACT. P. PREU and F. GUIDA, Arch. Neurol. & Psychiat. **38:** 818 (Oct.) 1937.

Four cases of psychosis following cataract extraction are reported. In 3 cases a diagnosis of psychogenic "experiential" panic was made. This condition is characterized by restlessness, apprehension, fear, "experiential" confusion and in some cases paranoid misinterpretation of environment. In the fourth case a diagnosis of senile arteriosclerotic

mental disorder was made. This condition is characterized by sensorial and intellectual defects, with fluctuating disorientation and impairment of memory but no striking emotional disorder.

In the first type of psychosis the operation, the subsequent care and especially the unfamiliar setting of darkness are all so out of the realm of the patient's past experience as to produce an "experiential" confused state. This usually comes on within the first twenty-four hours after operation and is relieved within forty-eight hours after the removal of bandages.

In the second, or organic, type of psychosis, the operation plays no etiologic role, and removal of the bandage has no effect.

The authors emphasize the importance of recognizing the second type preoperatively by some inquiry into the intellectual status of the patient, for example, by memory tests. Administration of sedatives usually increases the already present clouding of consciousness and so are contraindicated. Frequently a combination of both types of psychosis is encountered. The authors suggest familiarizing the patient with the procedure and surroundings preoperatively, and in some cases in which mental difficulty is definitely anticipated, they even suggest operating in the home if possible.

R. IRVINE.

CATARACT AND LATENT TETANY. M. MECCA, Ann. di ottal. e clin. ocul. **65:** 609 (Aug.) 1937.

A woman of 25 had noticed failing vision for two years. Falling of the hair and fragility of the nails had also been observed. Vision was reduced to 3/50 in the right eye and 1/10 in the left eye. Both lenses showed numerous white punctate, needle-shaped opacities and also a ring of larger round or oval opacities. The subcapsular layers of cortex and the nucleus were involved anteriorly and posteriorly, leaving a clear zone near the equator of each lens. Neurologic examination showed signs of latent tetany. The blood calcium was 9 mg. per hundred cubic centimeters and the potassium was 19 mg. After discission and corneal expression, the course was practically normal, with recovery of vision to 7/10. The literature on so-called endocrine cataract is reviewed, which indicates that bilateral progressive subcapsular cataracts of this type are most commonly due to a glandular imbalance involving especially the parathyroid glands.

S. R. GIFFORD.

CONCERNING CATARACT FORMATION AFTER REDUCTION OF WEIGHT WITH DINITRO-BODIES. T. HELMINEN, Acta ophth. **15:** 490, 1937.

The 5 cases of cataract reported here occurred in Finland. All the patients were otherwise healthy women below the usual age for cataract. Two had used dinitrophenol; 2 had used dinitroorthocresol, and 1 had used both drugs. The usual therapeutic dose was never exceeded, and the formation of cataract did not seem dependent on the amount of the drug taken. The interval between the completion of the reducing treatment and the full development of the cataracts varied from seven to fourteen months. Prodromal disturbances of accommodation occurred in 3 cases. The cataracts developed over a period of from three to seven months. In 4 cases secondary glaucoma occurred shortly before the extraction of the lenses.

There were no operative or postoperative complications. The author tried unsuccessfully to produce changes in the lens by feeding the drugs to rabbits.

O. P. PERKINS.

Lids

LICHEN PLANUS OF THE EYELIDS. H. E. MICHELSON and C. W. LAYMON, Arch. Dermat. & Syph. **37:** 27 (Jan.) 1938.

Some areas of the body seem immune to the invasion of certain diseases. This appears to be true of the eyelids in lichen planus. However, Michelson and Laymon saw 5 patients in whom the eyelids were unmistakably the seat of such lesions. Three types of lesions were observed: (1) the classic lilac-colored slightly delled papules with filigree scaling, associated with similar lesions on the body: (2) papules so arranged as to make the typical annular or small medallion-like plaques which are often seen on the glans penis. (patients with this type had other annular lesions on the body) and (3) some unique lesions. The third type was noted in 2 brunette women, aged 29 and 32. The upper eyelids were discolored with a sepia retiform eruption, which was not made up of elevated papules, although in the skein of the network were small knobs which could have been papules previously. The eruption caused no symptoms but gradually extended to all four lids and became darker, constituting a cosmetic deformity. In 1 of these cases a small bit of tissue was excised, and on microscopic examination it showed the characteristic bandlike infiltration of lichen planus. The rest of the skin and the mucous membranes were free from eruption in both cases. The eruption on the eyelids was identical with areas of melanotic staining seen in certain patients with lichen planus in the late stages of regression. This form of lichen planus of the eyelids could easily be ignored as a variant of that disease or might be diagnosed as erythema ab igne. Conjunctival lesions are identical with those seen on the buccal or on the genital mucosa.

J. A. M. A. (W. ZENTMAYER).

PALPEBRAL MANIFESTATIONS IN DELAYED HEREDITARY SYPHILIS. R. GOZBERK, Ann. d'ocul. **174:** 837 (Dec.) 1937.

Delayed hereditary syphilis often gives rise to such ocular lesions as keratitis, iritis, iridochoroiditis and neuritis, but its localization at the site of the lids is exceptional. Laurence reported a case in which signs of hereditary syphilis were present on the lids, lips and cheeks of a boy of 14 years. In 1888 Hutchinson described palpebral commissures, and von Michel in 1900 reported a gumma on the upper lid of a girl of 11 years. Other similar reports are recorded.

Delayed manifestations appear during childhood, adolescence and adult life. As a rule they develop spontaneously, but they sometimes appear during convalescence from infectious diseases or after trauma. Except in rare cases, these late appearances coexist with congenital dystrophies, which are of aid in making a diagnosis. Delayed hereditary syphilis may attack any part of the lid, skin, conjunctiva, subcutaneous tissue and tarsus. It appears in one of three forms: cutaneous gumma, subcutaneous gumma and gummatous infiltrations bordering on ulcer-

ation. The ulcerations may be so extensive as to bring about destruction of the lid.

Two cases are reported. The first was that of a boy of 11 years in whom a subcutaneous gumma developed in the parts surrounding the eye after an injury. In a few weeks an ulcerative process developed, causing irreparable mutilation of the lid. In the second case, that of a man of 30, syphilitic gummas appeared on the lid and on the face. In this patient, who was insufficiently treated, an injury revealed the latent syphilis. Two illustrations and a bibliography accompany the article.

S. H. McKee.

Methods of Examination

Ophthalmodynamometry and Its Importance in Clinical Medicine. T. R. Yanes, Rev. cubana de oto-neuro-oftal. **5:** 5 (Jan.-Feb.) 1936.

After a historical survey and a description of the technic of using the ophthalmodynamometer, the author stresses the importance of dividing patients with a high retinal arterial pressure into two groups, those with a coincident elevation of the general arterial pressure and those in whom the pressure is normal, and of studying in the latter group conditions of meningocephalic, ocular and vascular origin.

C. E. Finlay.

Measurements in the Fundus. W. Kühn, Arch. f. Ophth. **138:** 129 (Oct.) 1937.

With a heliometer built into the ophthalmoscope of Gullstrand (*Arch. f. Ophth.* **133:** 153, 1934), Kühn measured the width of the retinal vessels of 225 healthy persons and of a number of persons with vascular diseases and diseases of the optic nerve. In every case the micrometer scale of the heliometer was first standardized by measuring with it a fairly constant distance in the fundus, namely, the horizontal diameter of the disk; then the width of the vessels near the margin of the disk was measured in the thus standardized scale units. For healthy persons, the relation fo the caliber of the artery to the caliber of the vein and to the horizontal diameter of the disk was 1 : 1.2 : 14 to 16 (1 expressing the value for the arterial diameter). In absolute terms, the caliber of the artery varied from 0.086 to 0.138 mm. and that of the vein from 0.100 to 0.159 mm. In a case of occlusion of the central artery, the width of the latter was 0.03 mm., whereas the vein had a caliber of 0.125 mm.

P. C. Kronfeld.

Neurology

A Case of Epilepsy Associated with Meningioma of the Optic Nerve Sheath, Compressing the Olfactory Centers, Dural Calcifications and Thalamic Lesions. J. W. Papez and R. W. Rundles, Arch. Neurol. & Psychiat. **39:** 150 (Jan.) 1938.

The authors present a case of epilepsy with an olfactory aura in a physician who died at the age of 76 and summarize it as follows:

"Twenty years prior to death there developed retrobulbar optic neuritis on the right, which resulted in blindness. An olfactory aura appeared, and five years later epileptic attacks began. A meningioma of the right optic nerve sheath which compressed the basal olfactory centers was observed at autopsy. A tumor of the breast had been removed surgically. The adrenal glands were greatly enlarged. Degenerations of various fiber tracts caused by the meningioma, dural calcifications and lacunar softenings in the left thalamus and elsewhere are described."

R. IRVINE.

CONTRIBUTION TO STUDY OF BLINDNESS OF HYPOPHYSIAL ORIGIN: FOUR CASES OF ADIPOSOGENITAL SYNDROME WITH RETINAL DEGENERATION AND MENTAL BACKWARDNESS. P. PESME and G. HIRTZ, Gaz. méd. de France **44:** 833 (Oct. 15) 1937.

Pesme and Hirtz recently observed 4 cases of a curious hereditary and familial syndrome that is characterized by adiposogenital dystrophy with mental backwardness and retinal degeneration. The authors describe these cases not only because of their rarity but because they show the role of the hypophysis in the development of the syndrome and particularly the ocular lesions. The authors direct attention to the similarity of the disorder to the Laurence-Biedl syndrome. The described cases differ from those designated as the Laurence-Biedl syndrome by the absence of malformations of the members (syndactylism or polydactylism). There seem to be three symptoms that characterize the typical as well as the atypical cases: obesity of the hypophysial type, mental backwardness and retinal disorders. The retinal symptoms are especially noteworthy: In all cases there was a degeneration of the retina, with discoloration and night blindness. One of the patients was given extracts of the entire hypophysis for a year, but this form of treatment failed to produce the slightest improvement. After a new examination, it was decided to treat the patient with extracts of the anterior lobe of the hypophysis and with several other glandular preparations (thymus, thyroid, adrenal and whole pituitary). Under the influence of this treatment the patient lost weight and increased in height. Moreover, the genitalia developed to normal size; the hands and fingers, which had been extremely short, assumed a normal shape, and the nails lost their friability. The mental aspects likewise changed in that the boy became more active and bright. Even more remarkable was the change in the ocular defects. The boy was able to fix his vision without being disturbed by nystagmus, and the photophobia disappeared. Measurement of the visual acuity revealed hardly any increase, but the boy was better able to utilize his visual powers. On the basis of this observation the authors conclude that the hypophysis influences not only the fat metabolism and the development of the genital organs but the retina. Such a relation between the retina and the hypophysis had already been suggested by other investigators, and the authors think that treatment with hypophysial hormones may perhaps prove valuable in some congenital retinal defects.

J. A. M. A. (W. ZENTMAYER).

THE CHANGES IN THE EYE IN HERPES ZOSTER OPHTHALMICUS. A. N. MURZIN, Vestnik oftal. **11:** 758, 1937.

Three cases of herpes zoster ophthalmicus with serious complications are reported.

CASE 1.—A woman aged 30 had ocular changes consisting of diffuse corneal opacities in the superficial and deep layers, numerous retinal hemorrhages and marked inflammation of the optic disk, which gradually became atrophic, with resulting loss of vision. There was also paralysis of the abducens nerve on the side affected. Murzin believes that the herpes virus most likely penetrated the optic nerve and the retina through the sheaths of the ciliary nerves after having caused changes in the trigeminus nerve.

CASE 2.—A man aged 23 had herpes zoster of the region of the second and the third branch of the trigeminus nerve, keratoiritis and optic neuritis. Vision improved from 0.08 to 0.6 within one month.

CASE 3.—A man aged 63 had herpes zoster of the first branch of the trigeminus nerve with involvement of the sympathetic nerve, as Horner's syndrome was present. Murzin discusses the causation and the clinical and pathologic pictures of herpes zoster.

O. SITCHEVSKA.

Ocular Muscles

FUNDAMENTAL PLAN UNDERLYING OCULAR MOVEMENTS: I. THE CONJUGATE HORIZONTAL MOVEMENTS. J. OHM, Arch. f. Ophth. **138:** 1 (Oct.) 1937.

Summing up thirty years of his widely known work, Ohm gives a comprehensive presentation of his views concerning the innervation of ocular movements. At first the physical and the nervous mechanism of vestibular nystagmus are described. On slow rotation of a normal person in the turning chair in a dark room the eyes show no nystagmus but a tonic deviation in the same direction as the current of the endolymph which is caused by this rotation. On more rapid rotation, nystagmus with a slow and a quick phase occurs. The tonic deviation is independent of either phase of this nystagmus. Stimulation of the neuroepithelium of a single semicircular canal is sufficient to cause nystagmus. Nystagmus to the right, for instance, may be caused by an ampullopetal lymph current in the right horizontal semicircular canal or by an ampullofugal current in the left horizontal semicircular canal, by irrigation of the right external auditory meatus with hot water or of the left external auditory meatus with cold water and by destruction of the left labyrinth or of the left vestibular nerve. Ohm considers nystagmus with slow and quick movements as a process of discharge of energy by the vestibular nuclei which is not conditioned on the integrity of the vestibular nerves. In the state of rest, that is, in the state in which no strong stimuli are acting on the vestibular nuclei, there is a mutual exchange of energy between the two nuclei through intranuclear fibers until an equilibrium is reached. An ampullopetal lymph current increases the charge of the homolateral nucleus, whereas an ampullofugal current lowers the charge.

One of the cardinal points in Ohm's views is the assumption of a main stimuli-emitting station (*Hauptaugenmuskelsender*) for the ocular muscles which is located in the nucleus of the vestibular nerve. Even the impulses for voluntary ocular movements which are produced in the frontal center of gaze pass through the vestibular nuclei.

The pathways for optokinetic nystagmus and its disturbances are reviewed in great detail. The fiber tracts of the central nervous system which are concerned with ocular movements are graphically represented in three diagrams.

P. C. KRONFELD.

ESOPHORIA AND ANAPHORIA IN TWINS OF THE SAME SEX. J. STREBEL, Klin. Monatsbl. f. Augenh. **98:** 788 (June) 1937.

Twin sisters aged 18, of mirror-like resemblance, had eyes of identical appearance and muscular asthenopia. They complained of pain in the forehead, which disappeared when the hyperphoric right eye was covered for work at close range. Vertigo was absent. The eyeballs were free from pathologic changes. Slight esophoria, positive hyperphoria, anaphoria of 4 prism diopters in the right eye and of 3 in the left eye and homonymous double images of about from 10 to 12 prism diopters were found. As prism lenses had afforded only transient relief, recessions of the upper half of the medial rectus muscle, and of the nasal half of the superior rectus muscle were done under phorometric control. Complete disappearance of the disturbance resulted.

K. L. STOLL.

Orbit, Eyeball and Accessory Sinuses

DISLOCATION OF THE EYEBALL AS A COMPLICATION OF OXYCEPHALY. J. SHERNE, Brit. M. J. **1:** 565 (March 12) 1938.

A child 8 months old was first seen at the General Infirmary in Leeds on July 12, 1935, at which time there was marked protrusion of the forehead and exophthalmos was present. The roentgenographic appearances were typical of oxycephaly. The child was seen again at the age of 2 years. Vision was supposed to be defective. On April 8, 1937, the left eye became suddenly completely dislocated forward and was immobile. After anesthetization, the eye was gently levered back into position with a spatula, and to prevent a recurrence, a lateral tarsorrhaphy was done. Examination of the right fundus showed slight pallor of the disk. The child recovered promptly, and there has been no return of the dislocation.

In attempting to explain this condition, the author assumed that the proptosis was so marked that the action of the orbicularis muscle was largely nullified or reversed. In a fit of crying, the spasm of the orbicularis muscle was so great as to dislocate the globe.

ARNOLD KNAPP.

AN INTERESTING CASE OF THROMBOPHLEBITIS OF THE CAVERNOUS SINUS. A. FRANCES, Rev. oto-neuro-oftal. **12:** 79 (March) 1937.

A case of thrombophlebitis of the cavernous sinus of dental origin with a fatal outcome is reported. The author stresses the possibility of this origin, as it is usually overlooked.

C. E. FINLAY.

CLINICAL VALUE OF ROENTGENOGRAPHIC STUDIES OF THE OPTIC FORAMEN. B. J. FARBEROV, Ztschr. f. Augenh. **89:** 208 (June) 1936.

Roentgenographic studies were made of the optic foramens of 300 persons. The technic was not described in detail, but to be of any value identical procedures must be carried out for both the right and the left foramen. Pathologic conditions of positive clinical value were found in only 22 per cent of the cases. Normal roentgenograms were of definite negative clinical value in 6 per cent.

Of the pathologic conditions, enlargement of the canal was found in 17 cases. In 4 of these there was no general pathologic process to account for the enlargement. Unilateral unexplained papilledema was associated with enlargement in 1 case, which three years later showed no change. The enlargement in the other cases was caused by various types of tumors of the optic nerve and orbit.

Narrowing of the canal was found in 9 cases, caused by trauma, tumors, congenital atrophy of the optic nerve, Crouzon's disease and syphilis.

Deformities of the canal were noted in cases of congenital anomalies of the skull. General thickening of the wall of the canal was caused by osteoblastic metastatic tumor, congenital atrophy of the optic nerve, syphilis and hemangioma of the orbit. Thinning of the wall of the canal was caused by tumors of the optic nerve and fibromatosis of Recklinghausen. Farberov feels that roentgenographic studies should be obligatory in cases in which there are atrophy of the optic nerve, exophthalmos, retrobulbar neuritis, unilateral papilledema, trauma to the skull, and unusual atypical pathologic changes in the visual field or fundus.

H. GIFFORD JR.

Physiology

THE NATURE OF THE AQUEOUS HUMOR. J. D. ROBERTSON, Brit. J. Ophth. **22:** 79 (Feb.) 1938.

Robertson considers the several points raised by Duke-Elder in an article criticizing his paper concerning the theories on the formation and exit of the aqueous humor. He comes to the following conclusions:

The canal of Schlemm is not a vascular capillary in any sense, physiologic or anatomic.

Further study of the urea and the sugar equilibrium shows that the aqueous humor cannot be formed by dialysis because (*a*) the concentration of urea in the aqueous is lower than that in the blood and (*b*) the concentration of sugar in the aqueous is also lower.

As secretions and dialysates are both isotonic with blood, observations on the osmotic pressure are of no value in differentiating the one from the other.

A physical equilibrium does not exist between blood and aqueous, and the equilibrium level of the intraocular pressure is not maintained by the hydrostatic force in the capillaries minus the difference in osmotic pressure between blood and aqueous.

This is a further argument against the theory of dialysis. In summary, it may be stated that the observations which the author reported

originally refute the suggestion that the aqueous humor is a dialysate, and Duke-Elder's most recent contribution does not cause him to alter this view.

W. ZENTMAYER.

Retina and Optic Nerve

RETINAL MYELIN FIBERS. A. GARCÍA MIRANDA, Ann. d'ocul. **174:** 744 (Nov.) 1937.

Retinal conditions in man and in the higher animals present a great problem which experimental biologic research is endeavoring to solve. A study of embryologic and hereditary factors has helped to clear in an important manner this not thoroughly explored field. Among unexplained retinal conditions, one of the most interesting is that found represented by retinal myelin fibers.

The fibers were discovered by Virchow, who in 1856 first observed white patches in the neighborhood of the papilla. The year before Jaeger observed myelin fibers in the retina on ophthalmoscopic examination and had considered them to be an enlargement of the papilla. Nowadays numerous authors stress the familial orgin of this condition. Mauthner observed it in 2 sisters, and Kiso observed it 6 times in a family during two generations. Schieck concludes that it is a hereditary condition.

The ophthalmoscopic appearances are well known. In typical cases brilliant white patches are seen branching laterally above the papilla. At the periphery these have the appearance of silk thread; near the papilla they are more opaque and more prominent. The vessels appear in some parts to be covered by these; in others, they lie in the myelin fibers. The fibers rarely pass the borders of the papilla, but this varies considerably. Usually the fibers come just to the border without going beyond, appearing slightly opaque. Sometimes the papilla is affected in only a small degree, while at times it is completely involved, with the retinal vessels visible only in the retina. One illustration and a bibliography accompany the article.

S. H. MCKEE.

PSEUDO ALBUMINURIC RETINITIS IN CASES OF TUMOR OF THE BRAIN. I. J. MERKULOW, Acta ophth. **15:** 406, 1937.

Merkulow records his observations on 8 patients, all of whom on ophthalmoscopic examination showed the type of lesion in the fundus which is ordinarily associated with renal disease. One patient proved to have an abscess in the temporal lobe. The other patients all had tumors of the brain. Examination of the urine and blood and a test of the renal function eliminated involvement of the kidney in each case. In no case was the blood pressure elevated.

The author theorizes as to the mechanism of the production of pseudo albuminuric retinitis. He reviews briefly the classic theories as to the production of real albuminuric retinitis, paying most attention to the opinion of Volhard, who holds that the retinitis is not the result of hypertension as such but of the ischemia resulting from vascular changes. In the cases under consideration the author believes that the cycle of

events is as follows: Even in the absence of high systemic blood pressure there may be intracranial hypertension. This gives rise to a choked disk. The choked disk causes a disturbed retinal circulation, with eventual changes in the vascular structure and permeability. Deposition of fatty elements and the other changes of pseudo albuminuric retinitis result, but only if an additional factor is present. The additional factor is held to be a toxic product in some way elaborated by the tumor of the brain.

O. P. PERKINS.

Uvea

PATHOGENESIS, ETIOLOGY AND TREATMENT OF GLAUCOMATOUS IRIDOCYCLITIS. L. WEINGOTT, Arch. d'opht. **53**: 672 (Sept.) 1936.

In this paper the author offers an explanation for the occurrence of persistent hypertension in some cases of iritis. He reviews the work which has been done and the theories proposed to account for the secondary glaucoma which sometimes persists in spite of treatment. As a result of his observations, the author suggests that persistent hypertension in cases of iridocyclitis is due to degenerative changes which are commonly the result of syphilis and alcohol. He believes that alcohol increases microbic virulence and causes chronic anatomic changes. Inasmuch as gonococcic infection has been found in so many instances, the treatment he advocates includes the administration of vaccine in addition to the usual therapeutic procedures.

S. B. MARLOW.

Society Transactions

EDITED BY W. L. BENEDICT

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

CHARLES R. HEED, M.D., *Chairman*

Dec. 16, 1937

ALEXANDER G. FEWELL, M.D., *Clerk*

INTRACAPSULAR CATARACT EXTRACTION: REPORT OF A FURTHER SERIES OF CASES. DR. LEIGHTON F. APPLEMAN.

This article was published in full in the April issue of the ARCHIVES, page 548.

BILATERAL SUPERFICIAL LESIONS OF THE CORNEA DUE TO ALLERGY: REPORT OF A CASE. DR. GEORGE J. DUBLIN.

A white woman aged 48 complained of pain and inflammation of the eyes for one week. She had suffered a similar attack four years previously.

Vision in each eye equaled 6/6. Both eyes showed moderate bulbar injection; the corneal epithelium contained multiple punctate infiltrates, which were thickened and slightly elevated and stained with fluorescein. The eyegrounds were normal. The patient complained of severe discomfort to light.

Medical examination disclosed slight involvement of one of the sinuses and secondary anemia. The condition in the sinus cleared, but the pain and discomfort continued in spite of usual treatment. Tests for allergy were made, and the patient had a strong 4 + reaction to orris root. All other tests gave negative results.

The patient was advised to discontinue the use of cosmetics containing orris root. Without treatment, relief was instantaneous, and within twenty-four hours there were only 5 stained areas in the right cornea and 20 in the left cornea, whereas there had always been approximately from 100 to 200 such areas in each cornea for the past four months. The patient was seen frequently during the following three months, and in this time there was no recurrence of symptoms.

DISCUSSION

DR. WILLIAM ZENTMAYER: At the time I saw this patient the conjunctiva of the upper lids was quite papillary, and there was vascularity of the limbus together with the other changes in the corneas described by Dr. Dublin.

The condition somewhat resembled trachoma, and I thought that this possibility should be borne in mind, although there was also a

likelihood that it could be due to cosmetics. I think that Dr. Dublin has conclusively shown that the latter was correct.

DR. LEIGHTON F. APPLEMAN: I believe observations of this kind are of considerable importance. Occasionally one sees in hospital practice patients with inflammation involving the lids and conjunctiva which resist all usual forms of medication. I have at times been at a loss to know what medication to use next. I believe that many of these inflammations may be of allergic origin and that the patient should be subjected to the usual tests to determine this.

ALEXANDER G. FEWELL, M.D., *Chairman*

April 21, 1938

WARREN S. REESE, M.D., *Clerk*

MUSCLE SPASM CAUSED BY EYEGLASSES. DR. LEO F. MCANDREWS.

A man 41 years of age presents this interesting finding: As soon as he attempts to wear his glasses, a distinct, hard mass appears over the region of each mastoid.

Palpation indicates that the mass is evidently a contraction of the occipitofrontalis muscle.

If the patient persists in wearing the glasses, the entire right side of the head and right ear become painful, and the neck becomes stiff. Removal of the glasses or firm pressure on the bridge of the nose causes the mass and discomfort to disappear.

The condition has been present for about four years. Thyroidectomy was performed three months ago, but the patient is still nervous and emotionally unstable.

There is no anatomic explanation for this condition. The probable explanation is that it is a neurotic manifestation associated with the toxic goiter and can be cured by appropriate psychotherapy.

DISCUSSION

DR. WALTER I. LILLIE: It is my impression that this is a spastic condition. Its duration of four years is suggestive of a postencephalitic syndrome. When the man puts his glasses on he has marked twitching of the eyelids. In a case of postencephalitic syndrome, especially if changes in the respiratory tract are present, any sudden touch to any part of the body stops the attack. It would be interesting to follow this patient to see if a parkinsonian syndrome develops.

PALSY OF THE OCULAR MUSCLES IN A CASE OF TOXIC GOITER. DR. C. E. G. SHANNON.

This is a supplementary report on a case of toxic goiter complicated by palsy of the ocular muscles. A preliminary report was made at the meeting of the section in April 1937.

H. D., a man aged 47, was admitted to the Jefferson Hospital on April 2, 1936, with a diagnosis of exophthalmic goiter. His chief complaints were tremor of the hands, palpitation of the heart, bulging of the eyes, diplopia, excessive perspiration and loss of weight. The blood count was normal, and the Wassermann and Kahn reactions were negative. The basal metabolic rate was 45 plus. Motility of the larynx was normal, with no apparent narrowing of the tracheal airway.

Subtotal thyroidectomy was performed, and the patient was discharged ten days after the operation in good condition and relieved of all symptoms of toxic goiter aside from the proptosis and the diplopia.

Briefly, the ocular history of the patient is as follows: In March 1936, about a month prior to the removal of the thyroid gland, diplopia first developed; it was followed shortly by swollen and edematous lids and later by exophthalmos.

The diplopia and proptosis persisted, but the edema of the lids receded immediately after operation.

At the first examination of the patient, on Dec. 4, 1936, the following notes were made:

Vision in the right eye equaled 6/9 +; that in the left eye equaled 6/9 (mostly). The pupils were equal and reacted freely to direct and consensual stimulation. The tension was normal in each eye. The media and fundi were normal. The eyes were proptosed, the exophthalmometer showing 23 mm. of proptosis on each side. The left eye appeared definitely deviated inward and on a lower plane than the right eye. The Maddox rod test showed an esophoria of 30 degrees and left hypophoria of 26 degrees.

Diplopia was obtained in all the cardinal directions except in the immediate lower field, i. e., about from 12 to 14 inches (30 to 35 cm.) from the eyes, indicating involvement of all the extrinsic muscles of the eyeballs. It was interesting to note that a wide separation of images at 20 feet (609 cm.) diminished steadily as the light was brought toward the eyes, until at a comparatively near point the diplopia nearly disappeared, suggesting a paresis of divergence. The power of accommodation was affected. The patient read Jaeger's test type 14 at 4 inches (10 cm.) on the right side and at 5 inches (12 cm.) on the left side.

Various theories have been advanced in connection with the development of the exophthalmos, but so far none has proved entirely satisfactory.

The causation of palsy of the ocular muscles in cases of toxic goiter is still under discussion. Various theories have been presented by different authorities. When the preliminary report of this case was made an immediate operation to correct the muscular deviation was considered. In the discussion that followed Dr. Zentmayer suggested wisely that an operative procedure should be delayed until the congestion and the proptosis had further subsided. He cited the report by Naffsiger of cases of malignant exophthalmos with blindness in which enucleation was followed by death.

Two months later, with the patient under ether anesthesia, tucking of the superior rectus muscle and recession of the inferior rectus muscle were carried out. In addition, tucking of the external rectus muscle and the recession of the internal rectus were performed. There

was considerable reaction to these procedures, as might be expected, but with the use of ice compresses the eyes quieted, and binocular single vision was exhibited and still maintains.

CATARACT OPERATION TO REDUCE THE INCIDENCE OF PROLAPSE OF THE IRIS. DR. FRANK C. PARKER (by invitation).

This article will be published in full in a later issue of the ARCHIVES.

CLINICAL SIGNIFICANCE OF THE RETINAL CHANGES IN LEUKEMIA. DR. GLEN G. GIBSON.

This paper will be published in a later issue of the ARCHIVES.

EXTERNAL ORBITOTOMY. DR. EDMUND B. SPAETH.

(A motion picture in colors was presented, demonstrating the technic of orbitotomy by the external route without resection of the bone for the removal of retrobulbar tumor.) The tumor was in the muscle cone. Microscopic examination after its removal showed it to be a glioma of the nerve. In spite of the successful orbitotomy, it was thought wise in this case to do an enucleation for the satisfactory implantation of radium.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

JAMES W. WHITE, M.D., *Chairman*

Feb. 21, 1938

RUDOLF AEBLI, M.D., *Secretary*

SUPRASELLAR MENINGIOMA. DR. KAUFMAN SCHLIVEK.

A case of suprasellar meningioma is presented to demonstrate how proper visual fields, correctly interpreted, can make an elusive diagnosis straightforward.

L. K., a woman aged 51, complained of loss of vision in her left eye for one and one-half years. Aside from controlled diabetes and treatment for obesity, her past history was unimportant. Physical examination gave essentially negative results. Vision was reduced to 3/200 in the left eye; that in the right eye was 15/15. The field of vision of the left eye showed a temporal hemianopia and that of the right eye showed reduction of the upper temporal quadrant for color. Roentgenograms were normal. The patient had been treated for retrobulbar neuritis and was encouraged in the belief that the condition was improving. Other oculists suggested vascular disease and a macular pathologic process as the cause. However, on proper analysis of the case I concluded that the lesion was a suprasellar tumor. This was confirmed by encephalography and proved by successful operation.

The vision and the visual fields showed marked improvement. At the last examination, several months after operation, vision in the right eye was 15/15 and in left eye 15/30 + +, and the visual fields were practically completely restored.

TOTAL SYMBLEPHARON WITH PLASTIC REPAIR. DR. W. P. GRIFFEY (by invitation).

Anterior, or partial, symblepharon may be readily repaired by excising the adhesions from the globe and covering the denuded area with a sliding or pedunculated conjunctival graft. Posterior, or complete, symblepharon is a more difficult problem and at times is not correctable by surgical means.

Aside from facial disfigurement, symblepharon may give rise to loss of sight and interfere with the ocular movements.

The case reported here is one of posterior, or complete, symblepharon involving the right lower lid. Considerable loss of motion was observed in the visual field away from the adhesions. There were also some astigmatic changes in the affected eye.

Mr. J. O., a white man aged 45, suffered from a lime burn of the right eye in May 1937, while working with plaster. The eye was described at that time as showing a burn of the margins of the upper and lower lids, nasally. There was marked destruction of tissue about the lower canaliculus. The conjunctiva on the nasal side of the globe, inferiorly, and on the lower lid was destroyed. The cornea was ulcerated from 3 to 7 o'clock at the limbus and inward to beyond the center of the pupil.

Six months later, at which time the patient was operated on, a firm adhesion had formed between the lower lid and the eyeball. Fibrous tissue extended into the cornea from 3 to 5 o'clock peripherally and centrally to the margin of the pupillary area. The inferior punctum was drawn laterally to a point near the limbus.

The patient complained of a sensation of constant pulling on the eye and diplopia on gazing to the right. The diplopia was most annoying when he looked up and to the right. The vision in the right eye was 20/30 and in the left eye 20/20.

The adhesion was excised from the cornea, local anesthesia being used. Then, by means of scissors, the fibrous mass was separated from the globe well down to the lower fornix. The bare area on the globe was covered by means of sliding conjunctival flaps. A free conjunctival graft, 1.25 by 0.75 cm., was taken from the upper lid of the right eye and placed on the denuded inner surface of the lid. This graft was anchored in place by sutures, and a pressure dressing was applied. At the end of seven days the sutures were removed, and the eye was uncovered.

The cosmetic and functional results were satisfactory. The visual acuity was corrected to 20/20 by means of lenses.

SUBCONJUNCTIVAL CYST SIMULATING PROLAPSE OF THE LACRIMAL GLAND. DR. JAMES W. SMITH.

The patient was presented before this section in May 1929 with bilateral dislocation of the lacrimal glands. The glands were subse-

quently successfully replaced into the lacrimal fossae by operation. The technic employed was described in the 1933 *Transactions of the Section on Ophthalmology of the American Medical Association*, page 43.

Four months ago two small conjunctival cysts were seen along the border of the palpebral lobe of the left lacrimal gland. The cysts ruptured during an attempt at removal. They were found attached to the scar of operation performed in June 1929. Two months ago the left upper lid appeared full, and a firm, movable, almond-shaped mass was palpated in the temporal quarter, which could be replaced on pressure into the orbit. A diagnosis of recurrent prolapse of the lacrimal gland was made, and removal of the gland was advised.

A transverse section of the lid exposed a large cyst attached by a broad pedicle to scar tissue and the septum orbital just inferior to the lacrimal fossa. The lacrimal gland was found to be in normal position. Pathologic examination revealed that the cyst was probably formed from remnants of the lacrimal gland left behind at the time of operation eight years before. The original description of the replacement operation indicated that a small portion of the palpebral lobe contiguous with the fornix conjunctivae could not be freed and doubtlessly remained outside the fossa, with the resultant formation of a subconjunctival cyst.

SUBCONJUNCTIVAL SECTION OF THE DUCTULES OF THE LACRIMAL GLAND AS A CURE FOR EPIPHORA. DR. P. CHALMERS JAMESON, Brooklyn (by invitation).

Section of the ductules of the lacrimal gland in cases of epiphora is more advantageous than the more heroic operation of extirpation of the lacrimal glands. The postorbital region is not invaded, and satisfactory cutting off of the desired amount of secretion is obtained by a comparatively easy subconjunctival procedure. The anatomy should be visualized.

Whitnall states that the ductules seldom exceed a total of 12. Nearly all open separately along a line about 4 or 5 mm. above the upper convex border of the tarsal plate. (They open when the lid is everted.)

If these ductules are severed subconjunctivally in this region, communication between the glands and the conjunctival sac is disestablished. The product of the lacrimal gland, after sectioning, is discharged beneath the conjunctiva and does not enter the sac.

It must again be remembered that the region just described is not above, but below, the tarsal curve when the lid is doubly everted.

The technic for section of the ductules follows:

The lid is doubly everted by forceps. The fornix is thoroughly exposed and put on stretch. An incision 4 mm. long is made at the fornix of the outer canthus. Stevens' scissors are introduced in this opening, and a subconjunctival separation of the conjunctiva from its basal tissues is made, measuring about 7 mm. broad and 14 mm. long. This sections the ductules. The scissors can be seen through the translucent conjunctiva, and the secretion of the ductules sometimes can be seen to bulge the conjunctiva.

In making this separation, the distinctive landmark is the upper tarsal curve. The breadth of this conjunctival separation should extend from the curve to a line 7 or 8 mm. below.

The reaction is slight. Edema of the orbital tissues is not noticeable. The cheek on the side operated on swells slightly, showing that the secretion is discharging into the tissues. This rapidly disappears. The patient can go home immediately.

The after-treatment consists of ice compresses and sterilization of the sac, if there is any reaction.

DISCUSSION

DR. JAMES W. WHITE: I was fortunate enough to see one of the patients to whom Dr. Jameson referred. I saw the second eye operated on; the patient was so satisfied with the result in the first eye that he came back for an operation on the other eye. This was the second case reported by Dr. Jameson.

DR. LEWIS WEBB CRIGLER: I should like to ask if Dr. Jameson thinks that this procedure might be advantageous immediately after extirpation of the sac in cases of chronic dacrocystitis. It would be a more simple procedure than the Toti operation or one of its modifications.

DR. P. CHALMERS JAMESON: I think that this procedure might be used to an advantage in cases of chronic dacrocystitis; the only disadvantage would be in not knowing just how the secretion is going to react after the operation. I should be inclined to do the double procedure at the time of removal of the sac, possibly not on all the ductules. I think that one of the great advantages of this method is that one can grade it; one can section only half of the ductules, or less, by cutting down the area of operation.

DR. JULIUS WOLFF: May I ask whether Dr. Jameson thinks that possibly an external canthotomy might aid in everting the lid and making the operative area more accessible?

DR. P. CHALMERS JAMESON: I think that a canthotomy might be of help, but I do not think that it is necessary as the entire area of operation is so easily exposed.

DR. JAMES W. WHITE: That is my impression; I did this operation after seeing Dr. Jameson do it, and a canthotomy seems unnecessary.

OBSTETRIC OPHTHALMOLOGY. DR. DEWEY KATZ (by invitation).

(The subject was discussed in the light of the present knowledge under the headings of changes in the visual field, ophthalmologic aspects of the toxemias of pregnancy, retinal detachment in pregnancy, ophthalmologic indications for the termination of pregnancy and sterilization, birth injuries, ocular disease in the puerperium and lactation period and ocular disease of the fetus and of the newborn.)

DISCUSSION

DR. ALVIN J. B. TILLMAN: I hope you have all enjoyed Dr. Katz's paper as much as I have. I for one have been entirely unfamiliar with a great deal of what has been said here, and in discussion I shall confine myself to a few of the great number of topics covered by Dr. Katz. As an internist I have for several years been interested in the toxemias of pregnancy, and in the course of a study of these I have collected data

on a large number of cases in which a long follow-up study was made. For this meeting I went over the records of these cases from two points of view: first, for a description of the retinal findings and, second, for the results of follow-up study. I have been able to study 109 cases in which the patients were followed on the average of seven years, and of these, 67 per cent showed hypertension. Of 26 patients who showed no retinal damage during the toxemia, 43 per cent showed hypertension later. The entire series of cases was analyzed as to vascular change, edema of the disk and the presence of retinal hemorrhage or of exudate, and the following facts were noted: Of a total of 99 patients, 71.7 per cent of those who showed vascular change alone had hypertension later. Of those with edema of the disk, 63.8 per cent later showed hypertension after an average of six and one-half years. Of the patients with retinal hemorrhage and exudate, 85 per cent showed hypertension after a period of from seven to eight years.

In agreement with what has been said here, there were 16 patients who showed vascular change before the seventh month, and of these 16, all showed hypertension later, and 2 died four and four and one-half years, respectively, after the toxic pregnancy.

Thirteen patients in the series showed vascular change, marked retinal hemorrhage and exudate and edema of the disk, and after an average of from six to seven years later, none was normal and 1 had died. Of these 13 patients with toxemic pregnancies, 70 per cent had stillbirths; that is, 9 of the 13 had stillbirths, and the remaining 4 had premature deliveries with a dubious outcome for the babies. The correlation between this type of retinal picture and the poor, dubious or fatal outcome can be seen.

I have had 5 patients with retinal detachment, and in these, too, there was an extremely poor outcome. Three of the patients had stillbirths and 2 had premature deliveries, and during the follow-up study, which ranged from three to eight years, only 1 patient was found to be free from hypertensive disease. Four of the 5 patients showed severe hypertension; 1 was normal.

There is one point on which I differ from the ophthalmologist; that is, his advice to the obstetrician in the presence of retinal hemorrhage in the vomiting of pregnancy. I have seen 3 patients with this picture. Of these 3, 2 died and 1 recovered. One of the 2 who died had had the pregnancy terminated two days after the finding of the retinal picture. The other patient, as a result of what happened to the first patient, did not have the pregnancy terminated, and she died too. The third had the pregnancy terminated before the onset of retinal hemorrhage, but this developed a few hours later. The patient survived, but paraplegia developed, which lasted for several months, and I believe now, four years after this event, that she still shows evidence of it. In spite of the fact that vitamins B and C are used intravenously in the treatment of such patients, I am against allowing pregnancy to continue. Vitamins B and C have their place in the treatment of pernicious vomiting long before retinal hemorrhage takes place. The vitamins need time to act, and the immediate condition of this type of patient is critical. If in spite of this treatment retinal hemorrhage develops, the outcome may be unfavorable. A few months ago I had a patient of this type who was

treated in this manner and was permitted to continue with pregnancy. Two months after the decision, and in spite of adequate doses of vitamins, hemiplegia developed.

In summing up the few points I have made, I should like to say that as a result of this meeting I have added considerably to my knowledge in this small field of the toxemias of pregnancy. This knowledge should be helpful in formulating prognostications in this type of case. Judging by the cases that I have seen, hypertension will eventually develop in the patient with complete neuroretinitis, whereas only from 40 to 50 per cent of those with other types of neuroretinitis will have this sequel. It is therefore highly important to study the retina in the active phase of toxemia and in the process of evolution, though one may be tempted to forego this in the presence of severe illness of the patient in the effort not to disturb her. Still, in the interests of the outcome, retinal examination is important.

DR. FRANCES RICHMAN: Dr. Katz has thoroughly and concisely reviewed the various ophthalmologic conditions associated with pregnancy and has brought out the need in these conditions for consultation and cooperation between the ophthalmologist and the obstetrician.

In a consideration of the ophthalmologic conditions of the newborn due to birth stresses, to which I shall limit my discussion, it will be seen that cooperation between the two branches of medicine is especially important if the infant is to be assured of perfect visual acuity at birth.

In an ophthalmoscopic study of 1,500 newborn infants here and abroad, I have found their classification simplified by separating them into two groups: those whose eyes have apparently successfully withstood the stresses of birth and appear normal in all respects and those whose eyes have been unable to withstand the stresses of birth and therefore show ocular changes as a result. The term "birth injuries" in this connection is to be avoided. It has a connotation signifying trauma due either to accident or to inexperience and should be reserved for the extremely rare cases of such nature reported in the literature. On the other hand, the term "birth stresses" signifies all factors associated with pregnancy and birth which constitute a menace to the health of the mother or the child. Against these the careful obstetrician can take suitable measures, for they lend themselves to analysis and study.

Birth stresses affecting the eye are biochemical and mechanical, the former beginning with conception and operating incessantly until labor begins; the mechanical stresses then predominate until the child is born and the cord tied. The fetal nutriment conveyed by way of the maternal blood stream may at one time or another during gestation lack necessary elements or actually contain substances deleterious to the vitality of the ocular structures. Any impoverishment of the maternal blood, whether it be due to an avitaminosis, to a hidden focus of infection or to other factors which Dr. Katz has pointed out, may leave an imprint on the infant's eye that illy prepares it to cope with the innumerable mechanical stresses to which it is subjected during birth, be this ever so normal. The immediate effects produced by the interaction of both groups of stresses, passive and active, so far as they have affected the eyes of the infant, can be readily ascertained by ophthalmoscopic examination, the eye being unique among sense organs in that it yields its secrets to inspection.

The presence of hemorrhage in the fundus is the most common sign that an eye has been unable to withstand the stresses of birth. Hemorrhages are of various types and extent and location and can be classified according to the retinal layer which each involves. Bright red striate extravasations, evidently situated in the nerve fiber layer, are absorbed rapidly and leave no trace of their presence; hence the necessity for early examination, on the first or the second day. A preretinal type of hemorrhage is next in frequency. This is dark red and shiny and is well circumscribed and long lasting. At the summit of each collection of blood is seen the tiny reflex of the ophthalmoscopic light, which definitely shows that the hemorrhages lie in front of the retina; the inference is that in spite of appearance and duration they may have the least damaging effect. Diffusely round dull red hemorrhages situated in the layers deeper than the nerve fibers may be the ones having the greatest potential damaging power. A type of hemorrhage combining flame-shaped extremities and spindle-shaped white centers of serum is often seen.

Aside from these, other signs of stress are edema of the disk, retina and macula; stretching of the disk; ruptures of Descement's membrane, and subconjunctival hemorrhages.

A routine "eyeprint" of every newborn infant, as I have described it elsewhere, containing a detailed description of the normal as well as the pathologic conditions seen at birth, would enable the obstetrician to study his case in retrospect as to possible causes of stress and their avoidance in future; it would furnish ophthalmologists with information of extreme value in studying and eventually clarifying ophthalmologic problems of a controversial nature.

DR. MARTIN COHEN: In these cases of toxemia of pregnancy I am much interested in how long the high blood pressure had existed prior to the toxemia.

DR. ALVIN J. B. TILLMAN: It is impossible to say with any degree of accuracy. Some patients were admitted to the hospital for emergency measures and were seen for the first time when the hemorrhages were already present. No information as to antecedents could be obtained on these cases. I have more data on the 16 patients I mentioned who had vascular change prior to the seventh month, and I should say that about one half of this group had had hypertension over a variable period of time.

DR. DEWEY KATZ: I make no claim for originality or priority of any of the statements made this evening. My main purpose has been to try to bring some sort of order out of chaos in the field of obstetric ophthalmology and to plead for a closer cooperation between the ophthalmologist and the obstetrician.

Book Reviews

A Text-Book of Ophthalmic Operations. By Harold Grimsdale, M.B., F.R.C.S., Consulting Ophthalmic Surgeon, St. George's Hospital, London, and Elmore Brewerton, F.R.C.S., Consulting Ophthalmic Surgeon, Metropolitan Hospital, London. Third Edition. Price, $6. Cloth. Pp. 322, with 105 illustrations. Baltimore: William Wood & Company, 1937.

The third edition of "A Text-Book of Ophthalmic Operations" is larger in format than the second edition and less in pagination. There are 12 chapters, in which are discussed in rotation operations on the muscles, operations for ptosis, operations on the lids, operations on the conjunctiva, enucleation, operations on the lacrimal apparatus, operations for cataract, after-cataract and glaucoma, respectively, and operations on the cornea, the sclerotic and the iris and for the removal of foreign bodies.

The content has been well arranged. The grouping of procedures, not always easy to accomplish, has been carefully worked out, as has also the division of operations into their respective stages. The latter serves as an excellent simplified guide for the student.

The illustrations, with the eye blocked in by a border to represent the lids, are adopted in this edition as previously. Though this manner of presentation is striking and novel and may accentuate the picture portrayal, it does not add beauty to the volume.

The artist has done his work well. Being an artist, however, he has frequently introduced a large triangular corneal reflex, which in some instances may confuse the operative procedure for the student.

The anatomic and physiologic prefaces to the chapters are excellent, well thought out and simply described. They constitute one of the strong contributions to the book.

While the descriptive text is brief for the most part, it is clear, and every chapter is supplemented by a complete bibliography. Modesty on the part of the authors is probably the reason why so little of their personal work is portrayed or described. Comment also in regard to procedure is discreet, although in some cases pointed.

The presentation of a large number of operations without evaluation may leave the student confused.

The first chapter on muscles gives brief, but lucid, descriptions of various operations, among which are the newer procedures. It might be mentioned that two of the most popular operations of advancement and shortening are omitted—one, the Worth procedure (described in the previous edition), probably the most popular operation of this type in Great Britain, and the other, the Reese method (shortening), the most frequently practiced in Eastern America.

More space might have been given to the special corrective capabilities of advancement and recession, the degree of correction obtained by each and the specific value of each in its own sphere, with emphasis on the necessity of using the operation best suited to the type of case at hand. Correlated with this, one might wish there had been stressed the necessity of exact diagnosis based not on deviation alone but on a careful computa-

tion and investigation of underlying conditions before and after exposure of the field; this to include with deviation an estimation of the innervation, muscularization and fascial conditions in a given case.

In the second chapter on ptosis the student will find a goodly array of operations from which to choose.

The desire of the authors to eliminate largely historical matter, as mentioned in the preface, has, to a certain extent, been carried out. It has not been altogether accomplished, however, as this would be impossible in a well rounded volume. This may explain why 3 pages are devoted to a certain operation for ptosis of the skin. There is little actual benefit the student could accrue from the performance of this procedure, as the deformity left after such a plastic operation is usually worse than the defect.

In chapters III and IV 50 pages are devoted to operations on the lids. Guides to selection are made plainer by the excellent preface to this chapter. The operations are well, though briefly, described, and the illustrations are good.

In the section on conjunctival operations, stereotype procedures are portrayed. This type of work presents many problems, so that much can be left to the ingenuity of the operator.

The necessities for enucleation, with the varying technics, and also obliteration of the socket and opticociliary neurectomy are ably discussed. The technic of insertion of glass balls, implants, foreign material and paraffin, and the technic of exenteration are described.

The operative procedures dealing with the lacrimal apparatus are well selected. The technic of the extirpation of the lacrimal glands is described. The innocuous subconjunctival section of the ductules will probably render these more dangerous procedures unnecessary in the future.

Sixty-five pages are devoted to cataract. This chapter is well done and well balanced. Surgeons may find some omissions of their favorite procedures. The reviewer is much enamored with the bridge operations, which are not mentioned, and an important omission may be the through and through needling of Ziegler for juvenile cataract.

The closing chapters are devoted to the surgical treatment of glaucoma and to operations on the cornea, sclerotic and iris and for the removal of intraocular foreign bodies.

The preface to the chapter on glaucoma is most excellent. In this chapter the principles are briefly and simply described and cover the field.

A short but clear outline of the work done to date for retinal detachment is inserted toward the end where the work of Gonin, Guist, Larsson, Lindner, Šafář, Szily, Vogt and Weve is described. This brings the volume to a close.

As already stated, there are possibly some omissions on major subjects, and the surgeon-at-large may not find his favorite procedure or technic mentioned; but if this were done for all, it would take a library to hold the volumes.

The student will find a large range of operations briefly but well described. These can always be supplemented by a profuse and excellent bibliography.

P. CHALMERS JAMESON.

Untersuchungen über die Augenhöhlen des Menschen in verschiedenen Lebensaltern. By Per Pallin. Pp. 107, with 22 figures and 12 tables. Stockholm: Isaac Marcus Boktryckeri-Aktiebolag, 1937.

In his introduction Pallin states that the present study was undertaken because of the well known fact that a convergent strabismus of moderate or low degree often decreases or even disappears at about the age of 15 and that some patients who have been operated at an early age often eventually have divergent strabismus.

The author has measured 240 skulls of newly born infants, young children, older children and youths. As a control, the measurements of 200 adult skulls (100 of male and 100 of female origin) were used.

Among the measurements made were the inclination of the two orbits to each other, the angle between the outer walls of the orbits, the angle between the inner walls of the orbits, the depth of the orbit, the biorbital width, the interorbital width, the distance between the optic foramens and the distance between the midpoints of the orbital openings.

Each group of similar measurements has been treated statistically to obtain the mean, the mean deviation and the mean deviation of the mean, with a discussion of the extent to which the differences between the various means can be used as a basis for ascribing any changes noted to growth.

The author also reports the measurements of the orbits of 6 fetuses, which were 63, 86, 105, 185, 210 and 330 mm. in length.

In his summary Pallin states that starting about the middle of fetal life and continuing after birth there is a gradual turning forward of the large wing of the sphenoid bone. The depth of the orbit also increases, as does the distance between the midpoints of the orbital openings. The angle between the orbital axes becomes smaller, and there is a corresponding decrease in the angle between the axes of the muscle cones.

After late childhood there is no further increase in the depth of the orbit. At this time, owing to the relatively greater increase in the biorbital and the interorbital width, the midpoints of the orbital openings turn out more than the optic foramens do, so that there is an increase in the divergence of the orbital axes. This increased divergence, however, is slight when compared with the diminution of the biorbital angle which occurs after birth.

During further growth there is a further increase in the biorbital angle, which ceases at about the age of 20.

The biorbital angle has the following values: in late fetal life, 61 degrees; in newborn infants, 59.73 degrees; in young children, 49.98 degrees; in older children, 52.79 degrees; in youths, 52.90 degrees and in adults, 54.14 degrees. One must assume that there are corresponding changes in the angle between the axes of the muscle cones.

The medial orbital walls converge posteriorly before birth. Owing to the development of the ethmoid cells, which bulge more and more into the orbit, increasing the width of the interorbital region posteriorly, these walls become approximately parallel by late childhood and maintain this position in later life.

The external orbital walls also change their position. During the latter part of fetal life there is a turning forward of the orbital surfaces of the great wings of the sphenoid bone, which continues until late childhood. Also, the frontal processes of the malar bones gradually diverge anteriorly, with regard to their orbital surfaces, until adolescence, with a corresponding definite enlargement of the biorbital width.

Pallin has not only done a tremendous amount of work in measuring skulls and tabulating his results, but, from his statistical treatment of the data, he has also indicated which changes are significant, which are of questionable value and which are of no significance, at least for the number of skulls studied.

This is another one of the excellent monographs which have recently been issuing from the Scandinavian countries. The work was carried on at the University of Uppsala under the directions of Professors Frederik Berg and Erik Agduhr. In my opinion it is a valuable contribution to the developmental anatomy of the orbit, and it should be of great value to future workers in the same field.

W. F. DUGGAN.

Vitamine und Hormone in ihren Beziehungen zur Augenheilkunde. (Abhandlungen aus der Augenheilkunde und ihren Grenzgebieten, no. 25). By Prof. Dr. Kurt Wachholder, Prof. Dr. Werner Kyrieleis and Dozent Dr. Reinhard Braun, with an introduction by Prof. Dr. C. Behr. Price, Swiss francs 9:40. Pp. 80, with 21 illustrations. Berlin: S. Karger, 1938.

Three surveys which were presented in a symposium on vitamins and hormones and their relations to ophthalmology at the last meeting of the North-West German Ophthalmological Society are reprinted from *Zeitschrift für Augenheilkunde*. This publication for ophthalmologists is most timely in view of the importance of this research and of the rapid progress of the knowledge in this field. Wachholder's introductory survey is a basic study of the role played by vitamins and hormones in the animal economy as a whole. He calls attention to what Lumière has called the renaissance of humoral pathology and to how this has colored clinical study as well as laboratory research and experiment. While vitamins are correctly named, as they are nutritional elements essential to life, only one is, strictly speaking, an amine. The hormones, chemical messengers having no nutritional function, are concerned with the medication of neural impulses. The physical, neurologic and chemical nature of these reactions is analyzed and presented in detail and with admirable clarity. It is shown that vitamins are closely allied to enzymes and may act either as stabilizers or as catalyzers. They may be produced in the animal organism, or at least prepared in the form of provitamins, while substances which are practically hormones are found in plants. Carbonic acid, acetylcholine and a substance closely akin to epinephrine are general (*Allgemein*) hormones which can be isolated even from unicellular organisms. Kyrieleis discusses ocular symptoms in diseases of the glands of internal secretion. He notes the interdependence of the various members of the endocrine system and considers the factor of imbalance quite as important as hyperfunction or hypofunction of a single organ. The role of the vegetative nervous

system is also considered, as is that of the cerebrum, especially the midbrain. The last-named structure is considered at length and most instructively in connection with the ocular manifestations of hyperthyroidism, such as Graefe's, Stellwag's, Möbius' and Dalrymple's sign, in exophthalmic goiter and in other diseases, such as epidemic encephalitis, postencephalitic parkinsonism and myotonia congenita (Thomsen). A detailed study of the cause and pathomechanism of exophthalmos then follows. The occurrence and the characteristic form of cataract in cases of tetany, latent and manifest, as well as in cases of certain closely allied diseases, such as myotonia dystrophica, are described in detail. Acromegaly, Addison's disease, diabetes and the various dystrophies associated with pathologic changes and dysfunction of the pituitary gland are also described. Tumors of the hypophysis and their mechanical effect on the optic nerve and in the causation of increased intracranial tension and possible endocrine factors in corneal dystrophies and in certain syndromes, such as those which bear the names of Crouzon, Hand-Schüller-Christian, Friedreich and Wilson, are considered briefly. This survey contains a wealth of clinical information and data of biopathologic research. It is amply documented and beautifully and profusely illustrated. The third and concluding article on vitamins and the eye, by Braun, although short, is particularly valuable in that it takes up the thread of discussion from the generalizing introductory remarks of Wachholder's basic study and analyzes the role of the individual vitamins in various ocular conditions, physiologic as well as pathologic. This includes the subject of the intraocular production of vitamins, the chemism of visual purple and the factors of photosensitization and light adaptation. Braun lays stress on the importance of recognizing the deleterious action of vitamin imbalance, just as in a similar discrepancy affecting the endocrine system. The interrelations and reciprocal actions, while less thoroughly understood, are without much doubt of equal practical significance.

PERCY FRIDENBERG.

Stereoskopische Bilder für schielende Kinder. By Prof C. H. Sattler, Konigsberg. Third edition. Price, 6 marks. Fifty-four double plates. Stuttgart: Ferdinand Enke, 1937.

The first edition of these plates appeared in 1928 and the second edition in 1933 (reviewed in the October 1934 issue of the ARCHIVES, page 625). The present edition consists of 54 split cards for diagnosis and therapy.

In the accompanying book of instructions are given suitable questions for use with the various slides as well as the procedure to be followed in using the plates.

The appearance of a new edition testifies to the popularity of the Sattler stereoscopic pictures.

W. F. DUGGAN.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariate, 66 Boulevard Saint-Michel, Paris, 6e.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316, Rotterdam, Netherlands.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. Stewart Duke-Elder, 59 Harley St., London, W. 1.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.
Place: Plymouth. Time: July 20-22, 1938.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.
Place: Heidelberg. Time: July 4-6, 1938.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

Ophthalmological Society of the United Kingdom

President: Mr. Gordon M. Holmes, 9 Wimpole St., London, W. 1.
Secretary: Mr. H. B. Stallard, 35 Harley St., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 7-9, 1938.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian Str. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel-Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7è.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

TEL-AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby Str., Tel-Aviv.
Secretary: Dr. Sadger Max, 9 Bialik Str., Tel-Aviv.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: St. Louis.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Washington, D. C. Time: Oct. 9-14, 1938.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick Tooke, 1482 Mountain St., Montreal, Canada.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Gordon M. Byers, 1458 Mountain St., Montreal.
Secretary-Treasurer: Dr. Alexander E. MacDonald, 421 Medical Arts Bldg., Toronto.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. C. Gardner, 11 N. Main St., Fond du Lac.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. F. C. Cordes, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. W. Howe, 740 St. Helens Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501–7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Don M. Howell, Alma, Mich.
Secretary-Treasurer: Dr. Louis D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. R. A. Kelly, 304 N. Main St., Mitchell, S. D.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. John Hunter McRae, 26 Sheldon Ave., S. E., Grand Rapids.
Secretary-Treasurer: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. M. Harris, Johnstown.
Secretary-Treasurer: Dr. C. Wearne Beals, Weber Bldg., DuBois.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: Capitol Life Bldg., Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Charles T. Flynn, 41 Trumbull St., New Haven.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Grady E. Clay, 384 Peachtree St., N. E., Atlanta.
Secretary-Treasurer: Dr. J. Mason Baird, 511 Medical Arts Bldg., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. Francis E. Le Jeune, 632 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: Gulfport, Miss. Time: May 8, 1939.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. F. N. Smith, Grand Rapids Clinic, Grand Rapids.
Secretary: Dr. Dewey R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. Frank N. Knapp, 318 W. Superior St., Duluth.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Roy Grigg, Bozeman.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Norman W. Burritt, 30 Beechwood Rd., Summit.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: Atlantic City. Time: June 1939.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Algeron B. Reese, 73 E. 71st St., New York.
Secretary: Dr. Chester C. Cott, 333 Linwood Ave., Buffalo.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516-6th St., Valley City.
Place: Fargo. Time: May 1939.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. S. B. Fishburne, 1430 Marion St., Columbia.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.
Place: Columbia. Time: Nov. 1, 1938.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. Kate Savage Zerfoss, 165–8th Ave., N., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 805 Medical Arts Bldg., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.
Secretary-Treasurer: Dr. M. H. Williams, 30½ Franklin Rd., S. W., Roanoke.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. Andrew Rados, 31 Lincoln Park, Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. J. Mason Baird, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Fred M. Reese, 6 E. Eager St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. E. Clifford Place, 59 Livingston St., Brooklyn.
Secretary-Treasurer: Dr. Frank Mallon, 1135 Park Pl., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Oto-Laryngological Club

President: Dr. Clarence Engler, 2323 Prospect Ave., Cleveland.
Secretary: Dr. Fred Dixon, 2060 E. 9th St., Cleveland.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, Republic Bldg., Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Hugh G. Beatty, 150 E. Broad St., Columbus, Ohio.
Secretary-Treasurer: Dr. W. A. Stoutenborough, 21 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Lester H. Quinn, 4105 Live Oak, Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1066 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. A. Gough, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 806 Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Louis Daily, 1215 Walker Ave., Houston, Texas.
Secretary: Dr. Herbert H. Harris, 1004 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. J. K. Leasure, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to June.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. E. N. Robertson, Concordia, Kan.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Paul Nilsson, 211 Cherry Ave., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Clifford B. Walker, 427 W. 5th St., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Gaylord C. Hall, Brown Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Charles K. Beck, Starks Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Earle Breeding, 1801 I St., N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. John E. Mulsow, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. P. H. Kilbourne, Fidelity Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., monthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. L. de G. Joubert, 690 Dunlop St., Montreal, Canada.
Secretary: Dr. K. B. Johnston, 1509 Sherbrooke St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Guy Maness, 119–7th Ave., Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Percy Fridenberg, 38 W. 59th St., New York.
Secretary: Dr. David Alperin, 889 Park Place, Brooklyn.
Place: Squibb Hall, 745-5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. Philip Romonek, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Ophthalmological Society of the University of Pittsburgh

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

Passaic-Bergen Ophthalmological Club

President: Dr. R. N. Berke, 430 Union St., Hackensack, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Walter I. Lillie 3701 N. Broad St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. N. H. Turner, 200 E. Franklin St., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Roy E. Mason, Frisco Bldg., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Russell Fletcher, 490 Post St., San Francisco.
Secretary: Dr. Avery Morley Hicks, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. R. Kirkpatrick, 6th and Walnut Sts., Texarkana, Ark.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. P. B. Greene, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. O. M. Rott, 421 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. James F. Cahill, 428 S. Salina St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. A. Lloyd Morgan, Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. R. F. Luke, Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queen's Pk. Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 I St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.